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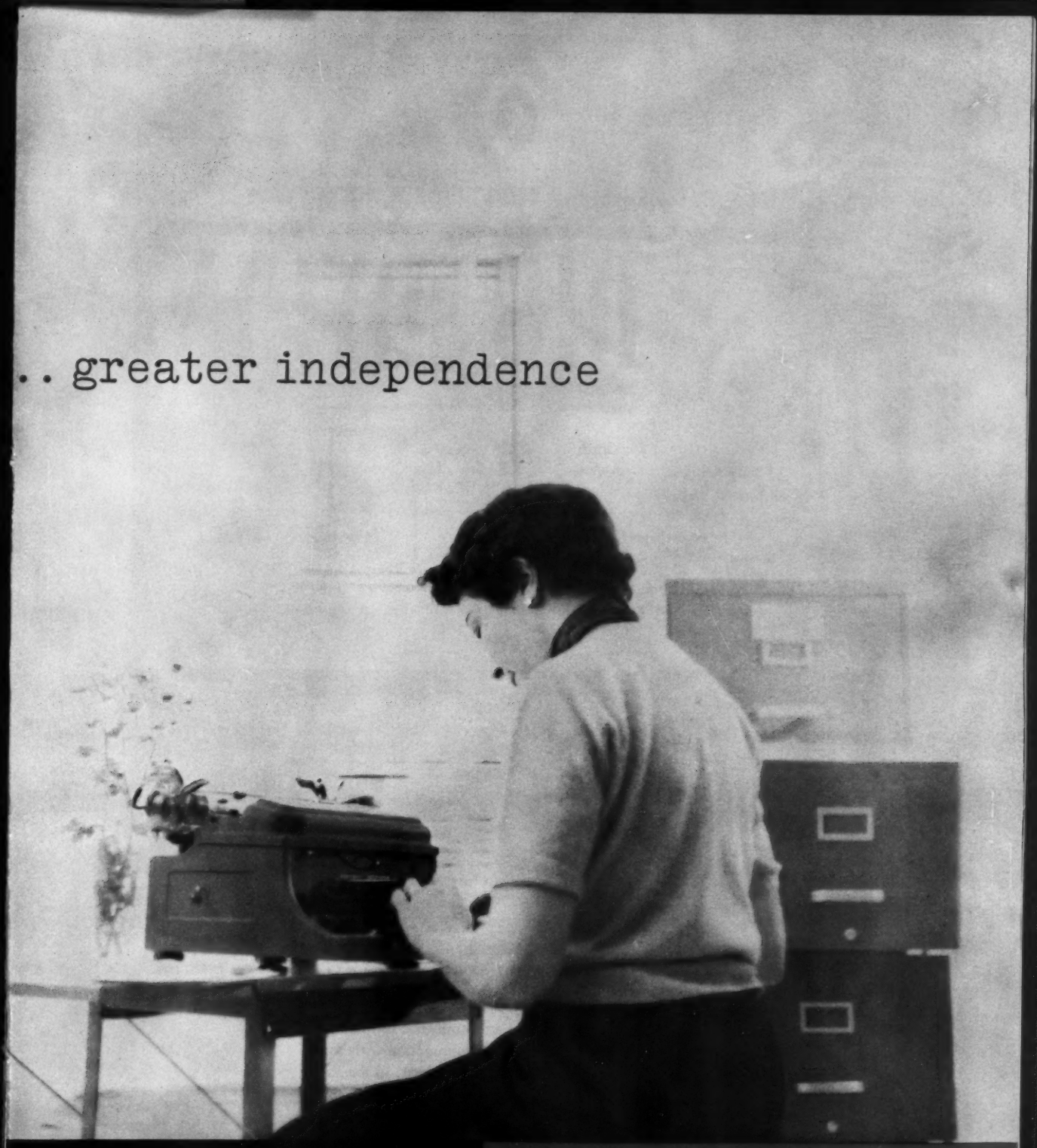

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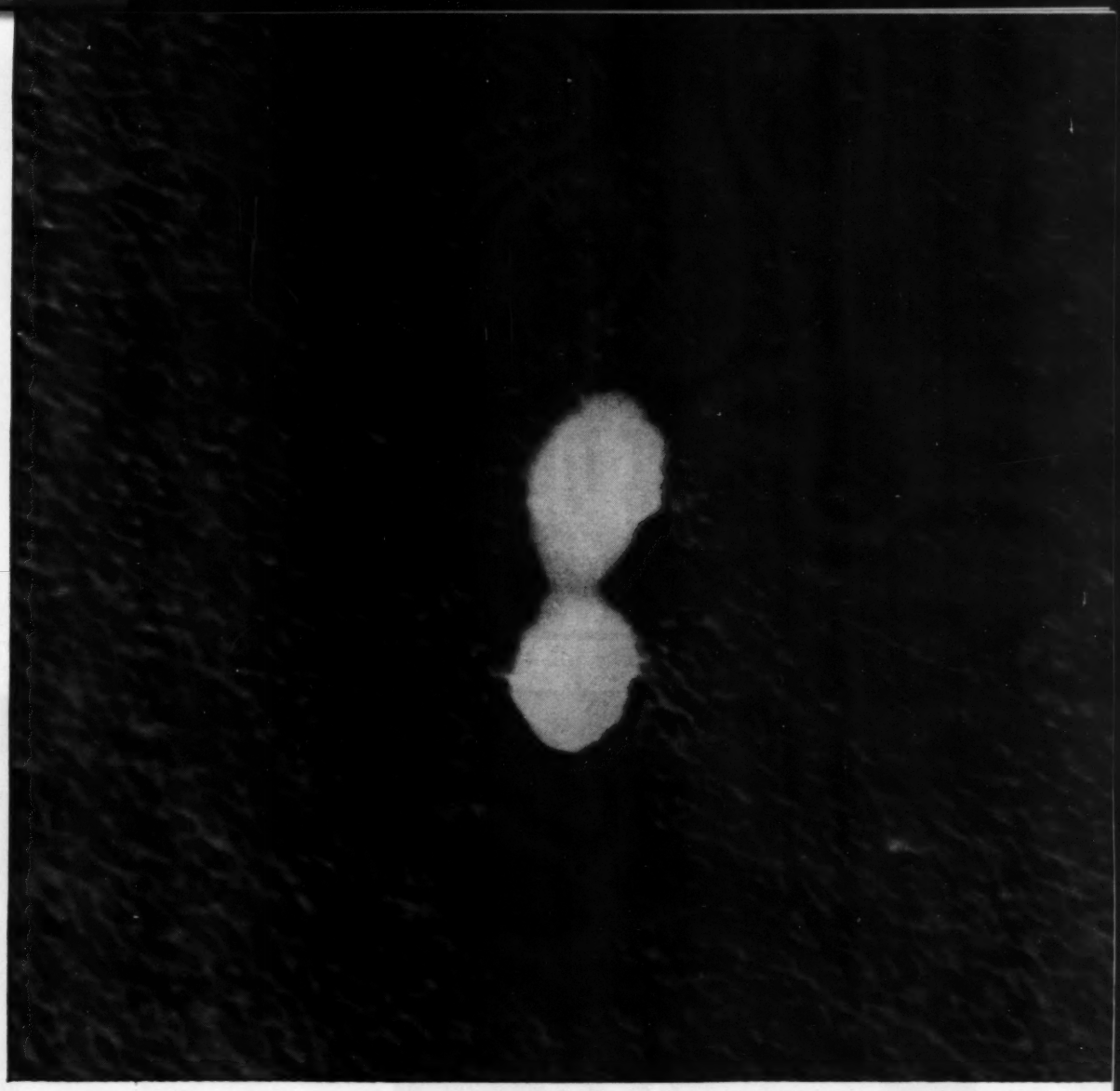

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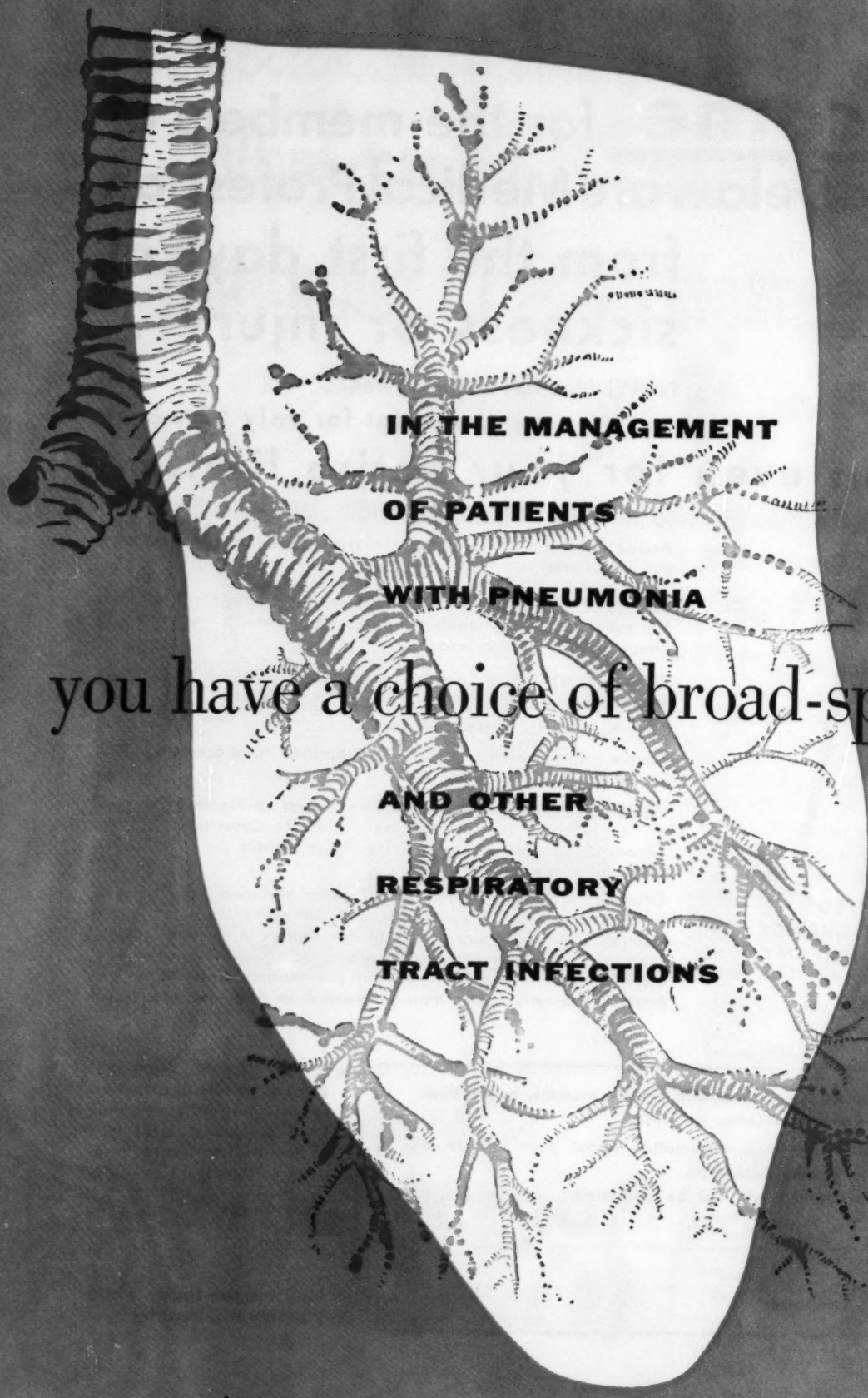

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1. *O'Regan, C., and Schwarzer, S.: J. Pediat. 44:172 (Feb.) 1954.*
2. *Waddington, W. S.; Bergy, G. G.; Nielsen, R. L., and Kirby, W. M. M.: Am. J. M. Sc. 228:164 (Aug.) 1954.*

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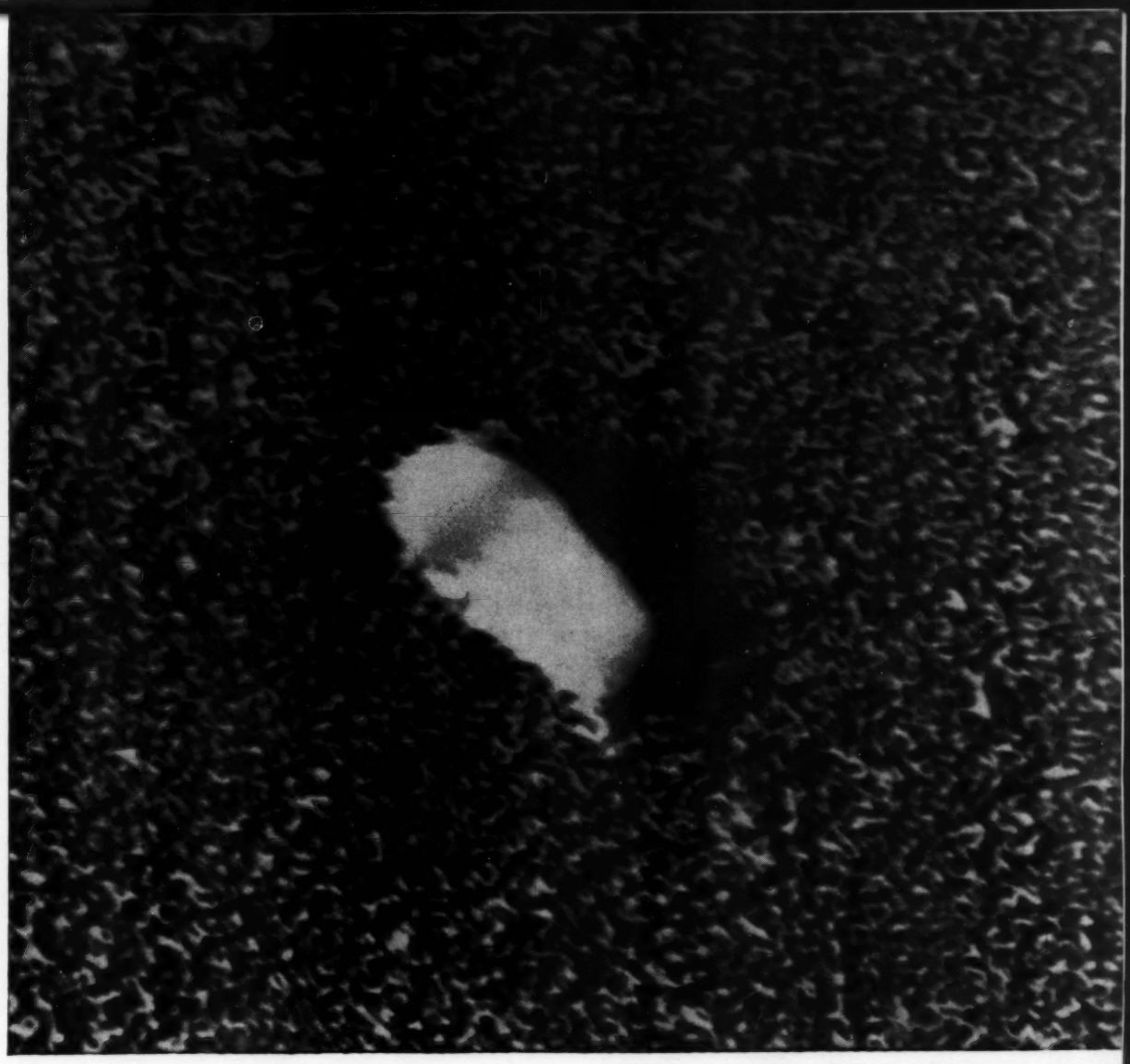

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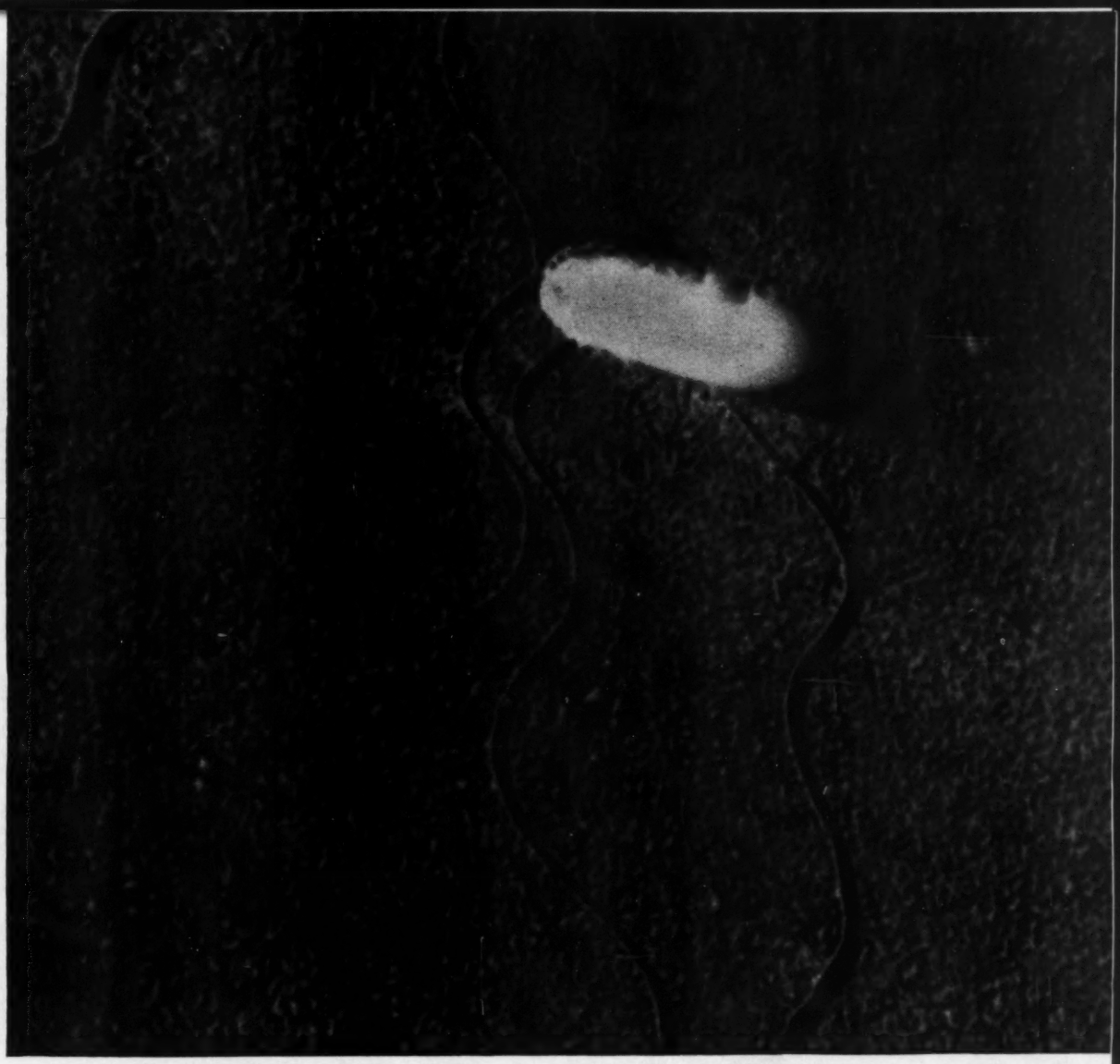

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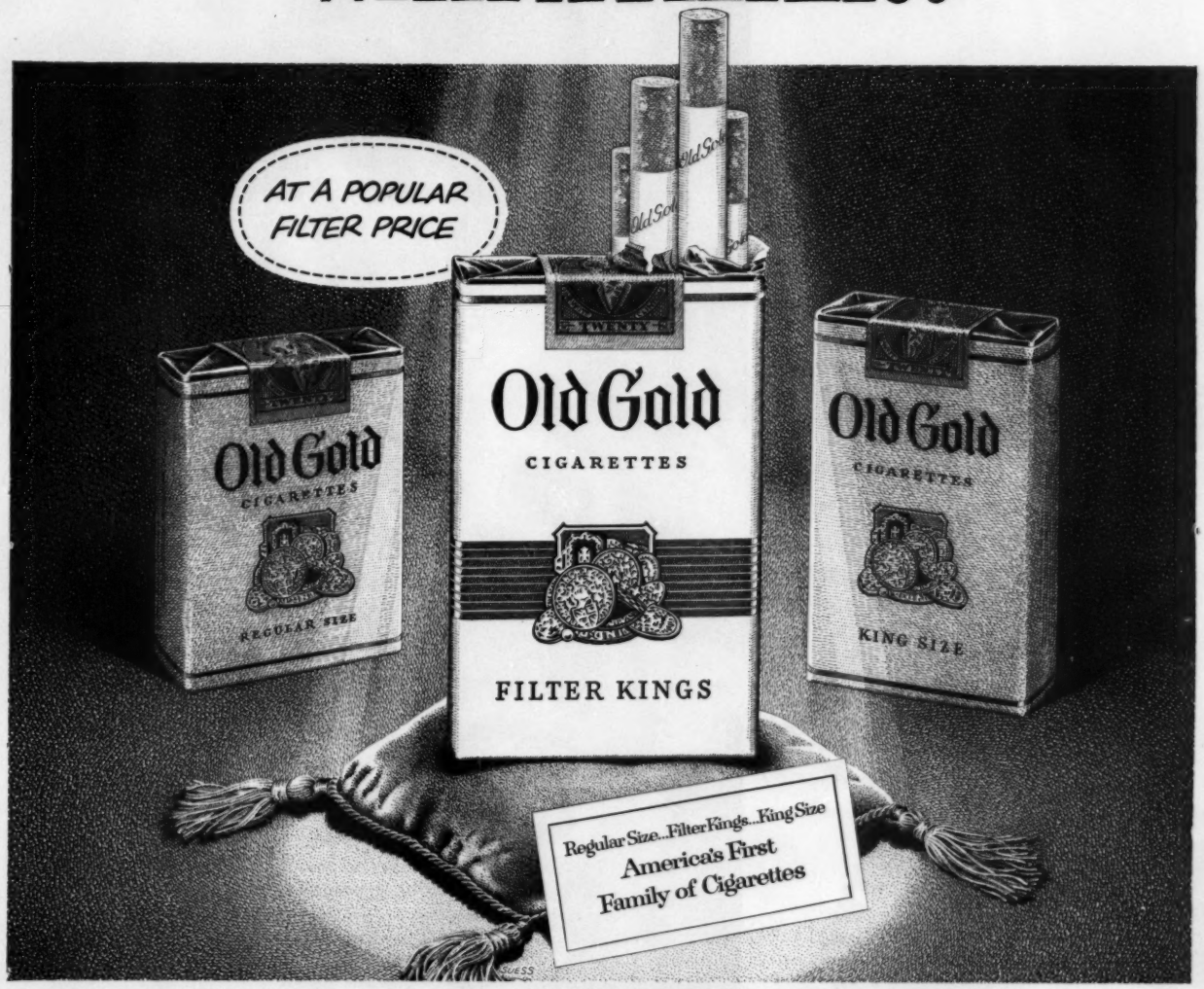

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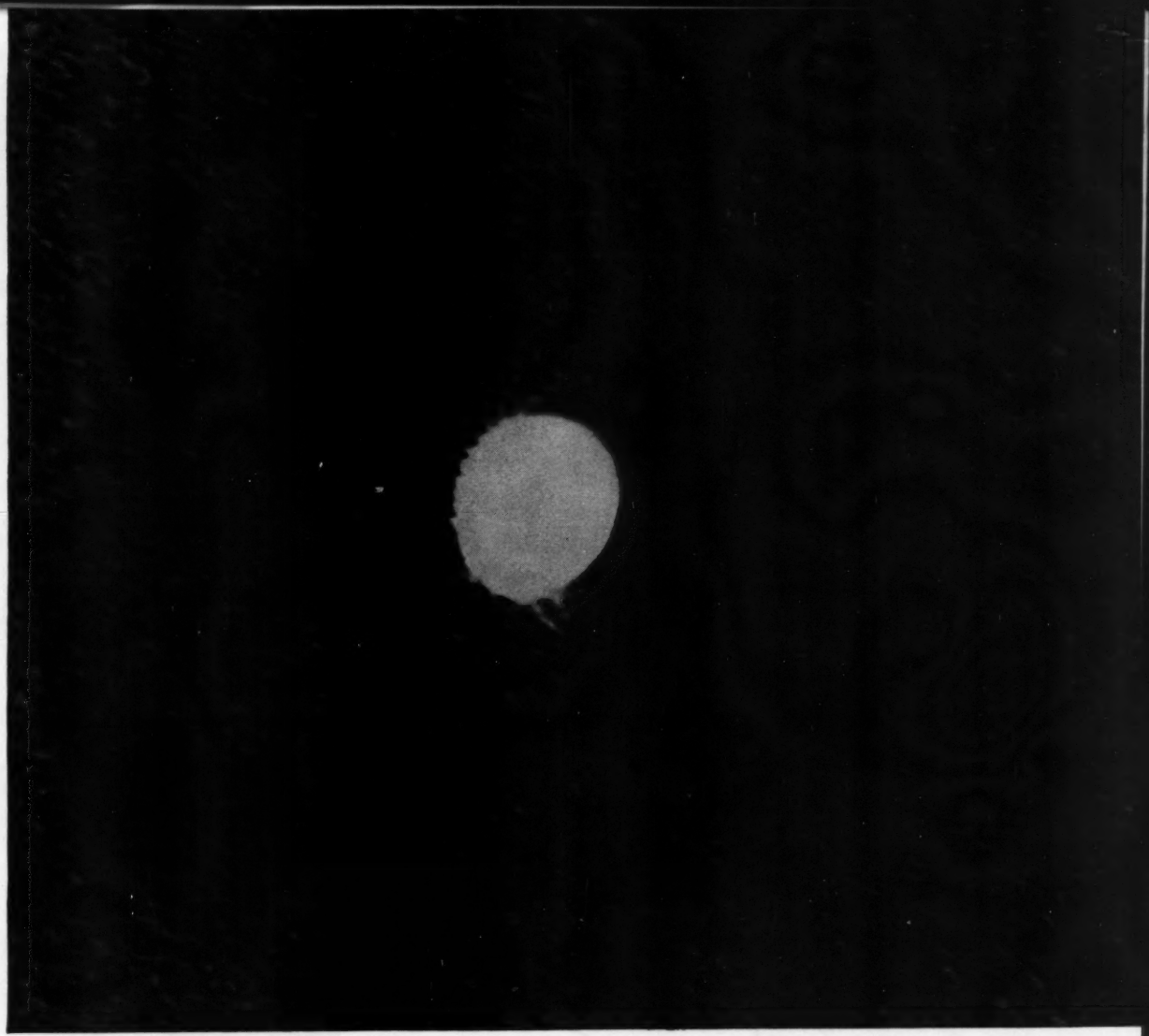

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Staphylococcus aureus 44,000 X

Staphylococcus aureus (Micrococcus pyogenes var. aureus) is a Gram-positive organism commonly involved in a great variety of pathologic conditions, including

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IMMEDIATE RECONSTRUCTION OF MAJOR TRAUMA TO THE HAND

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Wilmington, Del.

As the primary prehensile end-organ of the human skeleton, the hand is unique in its functions and complexities, and forms the primary outrigger of the corpus per se. Although completely normal function of the hand is dependent upon elasticity of the skin, proper alignment of bones, flexibility of joints, free play of tendons, and integrity of nerves, even a serious compromise of one or several of these functions can still yield a remainder which may be exceptionally facile. The role of the reconstructive surgeon is to evaluate the results of immediate or delayed major trauma with a view to reclamation of certain basic functions of the hand as a unit.

Although exceptional in its every aspect, the hand of the human being is perhaps most remarkable as a mechanical integral between its intrinsic parts and its parent limb.[3,12] This can perhaps be best characterized by the normal *attitude* or essential *balance* of the hand, and can be noted in almost every act. The wrist is stablized in moderate extension and slight ulnar deviation, as this is the most favorable position for the long flexors to act upon the thumb and fingers. The thumb is in direct alignment with the radius, with its metacarpal at right angles to the palmar plane. All fingers are semiflexed so that the distance from the finger pads to the thumb pad is equal, as is shown in Fig. 1, C. When brought into power, the functional analogy between the hand and a mechanical crane or shovel is valid, and in consideration of this analogy, the first law of the hand is realized: distal activity depends upon proximal stability. The wrist cannot act without stabilization of the elbow, the fingers cannot act without stabilization of the wrist, etc. Every reconstructive procedure must accept this relation as a primary goal and must be directed toward maintaining it, if present, or reclaiming it, if lost. Fig. 2 shows the late sequelae and end result of prolonged loss of balance during the reconstruction of major trauma. In this hand the wrist has stiffened in flexion, the metacarpal arch has contracted, and the thumb has been lateralized to the finger position. These factors, together with inadequate palmar resurfacing, have rendered the hand essentially useless.

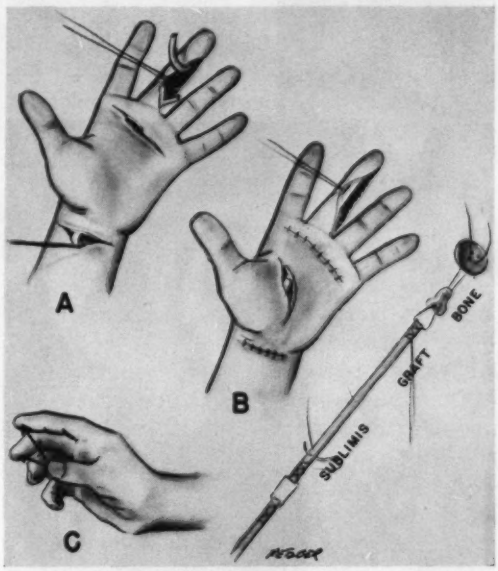

FIG. 1
(C) shows the normal attitude and balance of the hand; the finger and thumb pads are equidistant. (A) and (B) illustrates the method of inserting the sublimis tendon as a free profundus tendon graft.

Even though the pentadactyl hand-form is not unique to the human being, it is he who exposes it, more than any mammal, to the forces of trauma (Fig. 3). In studying specific examples of major injury and their repair, certain general technical considerations must be remembered. Surgery of the traumatic hand is usually emer-

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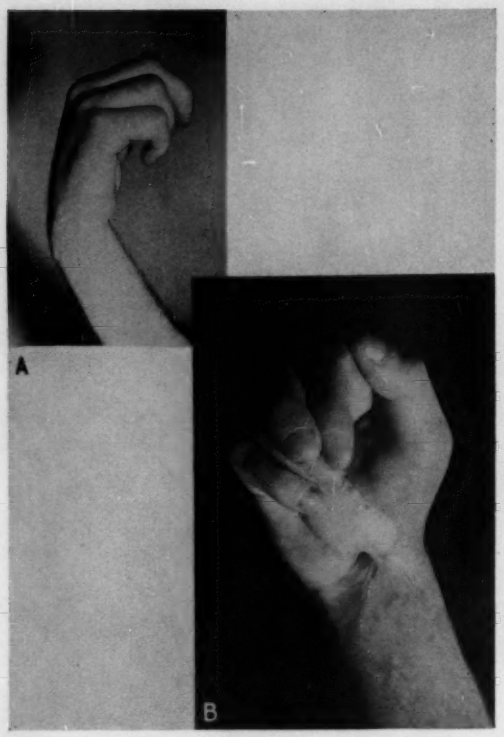

FIG. 2
(A) and (B) show the late results of maintaining the hand in poor position during prolonged reconstruction. The wrist is bound in flexion, the metacarpal arch architecture has been lost, and the thumb has been lateralized to the finger position. As a result, the hand has been rendered essentially useless.

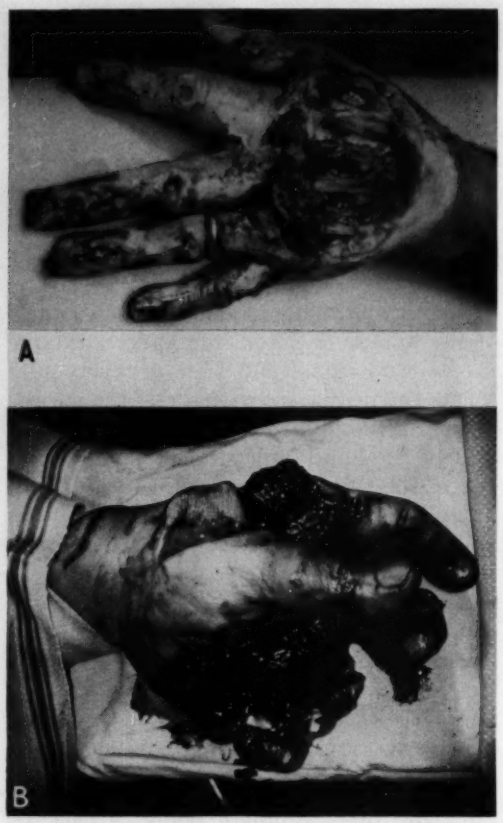

FIG. 3
(A) and (B) are examples of common major industrial trauma requiring extensive reconstructive procedures.

gent; however, the decision as to whether to close a wound or leave it open at the initial procedure is made largely on the judgment as to the chance of serious infection. Experience prior to the advent of broad spectrum antibiotics reliably demonstrated the danger in closing contaminated wounds after eight hours from the time of injury. Today, however, this concept must be revised, and a considerable gamble of the forces of infection against the tremendous advantage of immediate reconstruction can be taken.[11] Although the effect of these medicines on the destructive power of bacteria is striking, they will not prevent infection in poorly treated wounds. The basic principles of first-aid, tetanus prophylaxis, and meticulous preparation of the wound become of increasing importance as the rigid 'time-from-injury' rules are relaxed. In diagnosing infection in the posttraumatic hand, particular attention must be paid to the systemic signs of sepsis, for exudation of apparently purulent material from a badly damaged hand may represent only the products of decomposition of nonviable tissue, and may dissuade the surgeon from attacking his problem at its most opportune moment. Immeasurable gains in final function are attained by producing a reconstructive result before the ravages of edema, fixation, and fibrosis have become established.

The three objectives of reconstructive surgery are closure of wounds, reduction of fractures, and maintainence of attitude and balance. All procedures should be done under arterial torniquet.[1,2,3,4,8] Using the pneumatic blood pressure type of cuff, the extremity should be elevated and milked of its blood with a rubber bandage. With the newer Freon torniquet, elevation of the hand for a few moments prior to the instantaneous inflation of the cuff is suffi-

cient. Optimum pressure for the adult upper extremity is 280 mm Hg., and the cuff may remain inflated without fear of constriction paralysis for as long as four hours. Pressures above 280 mm Hg. are to be avoided.[6] Suture material for ligatures, subcuticular closure and muscle approximation should be 4-0 plain surgical gut. The use of steel wires for this purpose is to invite infection. In the treatment of damaged soft tissue a paradox of conservatism and radicalness exists. Sacrifice of any truly viable skin, fat, muscle, nerve, or tendon merely for the sake of converting a contaminated wound into a clean one is not justified; and yet at the same time failure to appreciate the true extent of the resurfacing problem will prolong the eventual disability many fold.[1,2,4,9,10] It is truly difficult at this stage to determine whether or not damaged tissue will survive. Errors in this respect concern mainly skin flaps and badly damaged digits.[11] Tendinous, nervous, ligamentous, and joint structures can usually be assumed to have a survival potential and their initial sacrifice is usually not wise. Neither is it wise to retain a digit from which the neurovascular pedicle has been destroyed. Likewise sacrifice of questionably viable skin and replacement by tissue of adequate blood supply is recommended.

The most common of wounds not in the crush-avulsion-blast group involve the tendons. Divisions of the extensor mechanism offer few problems except when at or distal to the metacarpal-phalangeal joint. Mason[7] has shown that the tendon does not actually divide into three bands at the MP joint, but rather is continuous fan-like structure with thin interruptions and a more or less condensed central portion with spread out lateral bands. The tendon actually inserts over the entire dorsal capsule of the proximal interphalangeal joint. Traumatic divisions are therefore never discrete as with the flexor tendons, but always involve the dorsal fascia and are associated with interruption of the dorsal interosseous attachment. Repair to attain maximum gliding function demands a meticulous diagnosis[12] of the extent of injury not only to the tendon but to the dorsal capsular mechanism as well. If completely repaired, gliding can be expected but even in this respect one is often disappointed. Rupture of the distal attachment can be repaired by hyperextension of the distal joint and flexion of the middle joint to 60 degrees. Position must be held four to six weeks and requires either plaster or Kirschner wire fixation. Late repair of the hammer deformity may require a short tendon graft due to contracture of the extensor hood.

Divided flexor tendons become a problem only when severed distal to the level of attachment of the lumbrical muscles. The profundus is likewise repaired without difficulty when severed distal to the attachment of the sublimis tendon. Repairs in the 'No-Man's Land' of Bunnell are most effective when the sublimis is withdrawn and reinserted as a free profundus tendon graft. The drawings in Fig. 1, A and B show a palmar laceration in which both tendons have been divided. The sublimis and the distal portion of the profundus are divided from their attachments through a midlateral finger incision. Fig. 4, A shows a method of determining the midlateral finger line. The sublimis is identified at the wrist and is withdrawn. This tendon is then reanastamosed to the profundus at the level of attachment of the lumbrical muscles and the suture line is buried in the soft tissue of the palm. The graft is then threaded through the profundus tunnels to the distal attachment. Fig. 5 shows the end result of such a procedure. The hand is dressed in the position of function and immobilized for three weeks. The pull-out wire technique is most convenient for the distal attachment, but has been largely given up for the proximal anastamosis.

Surgical repair of injured tendons requires adequate exposure. To attempt repair of these injuries using the wound of trauma alone is usually impractical. The hand can be freely incised without fear if the basic principle of the *working surfaces*[6] and their avoidance is remembered. Midlateral finger incisions should always be on the ulnar side of digits 2, 3, and 4,

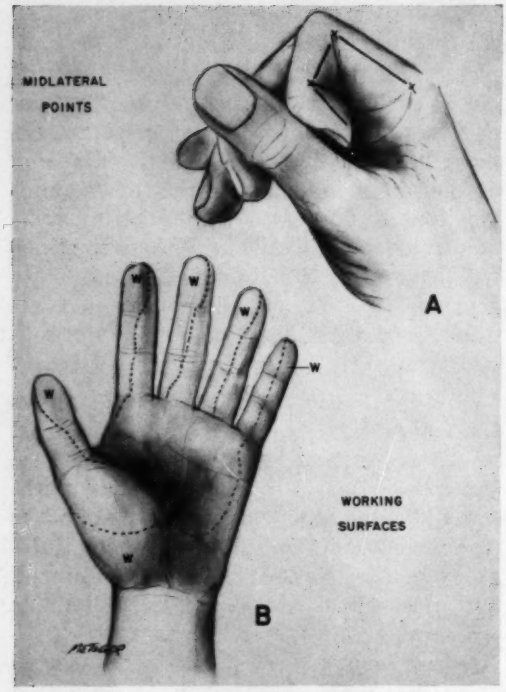

FIG. 4

(A) shows an easy method of determining the midlateral finger line by connecting the apices of the finger folds. An incision made in this line is not subject to contraction. (B) illustrates the working areas of the hand. Elective incisions in these regions must be avoided.

but on the radial side of the 5th. The working surface of the thumb is radial up to the MP joint and then becomes ulnar from there outward along the surface where the thumb and fingers oppose. Fig. 4, B illustrates the working surfaces. Palmar incisions should parallel flexion creases but not lie directly in them.

In treatment of the crush-avulsion-blast type of wound, reduction of fractures under direct vision is usually possible, although a preliminary x-ray study is essential. Removal of small fragments is accomplished and fixation of fractures by Kirschner wires with cross stabilization to adjacent bones can be done prior to definitive resurfacing. Final wound closure is usually done by a combination of the free graft, rotated local flap, and direct transfer abdominal flap.[10] The palmar surface of the hand is not adaptable to the use of rotated local flaps because of the septal attachment of the skin to the palmar fascia. On the dorsum the situation is more ideal. Loss of rotated flaps in this region is usually due to irreversable loss of venous drainage due to the trauma itself, or failure of the surgeon to preserve these veins when cutting and advancing the flap. In areas where denuded bone is not exposed and tendon sheaths are intact the heavy free split-thickness graft can be used to advantage. Losses in this type of coverage are due to an incorrect estimation of the viability of recipient bed, improper postoperative immobilization, hematoma formation, or infection. In general more grafts are lost by not looking at them soon enough rather than by the slight disturbing motions of a dressing

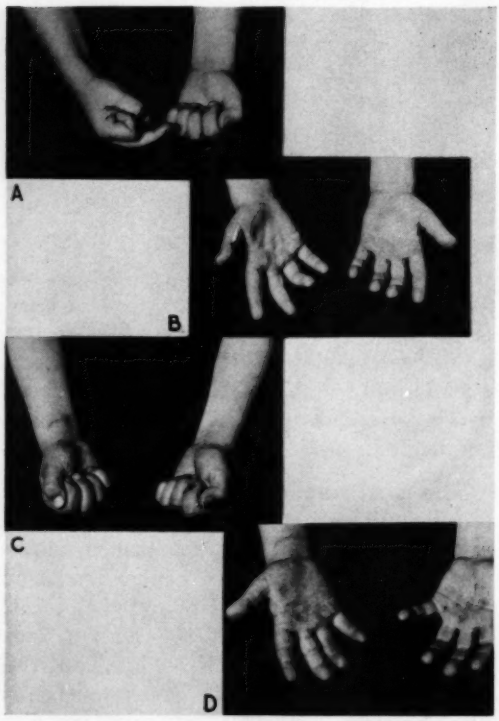

FIG. 5

Both tendons of the third finger of the right hand were divided at the distal palmar crease. Primary repair of both tendons was unsuccessful, as was a secondary attempt, (A) and (B). Insertion of the sublimis as a free profundus graft has allowed free gliding with normal dexterity of the finger, (C) and (D). This could have been accomplished at the initial procedure.

change. A careful change of dressing as early as the third postoperative day may give the surgeon a chance to reclaim a potentially lost graft by evacuation of hematoma. A graft that is not examined

for 7 to 10 days will be lost should any complicating factors have taken place.

In resurfacing losses with exposed bone, joints, or damaged tendons a full thickness skin flap is required. When a local rotated flap cannot be used a subcostal or epigastric abdominal flap is the tissue of choice. Blood supply in the subcostal region is insured by the internal mammary, long thoracic, and perforating intercostal vessels. The blood supply of the epigastric region is supplied by superficial branches of the deep epigastric arteries. In neither region should the survival of a correctly cut flap be questioned. In general, the flaps should be cut quite thin, and their length should not exceed twice their pedicle width.

The subcostal flap is used in resurfacing volar defects, while the epigastric flap is adaptable to closure of dorsal wounds. Examples are shown in Fig. 6. Perhaps the

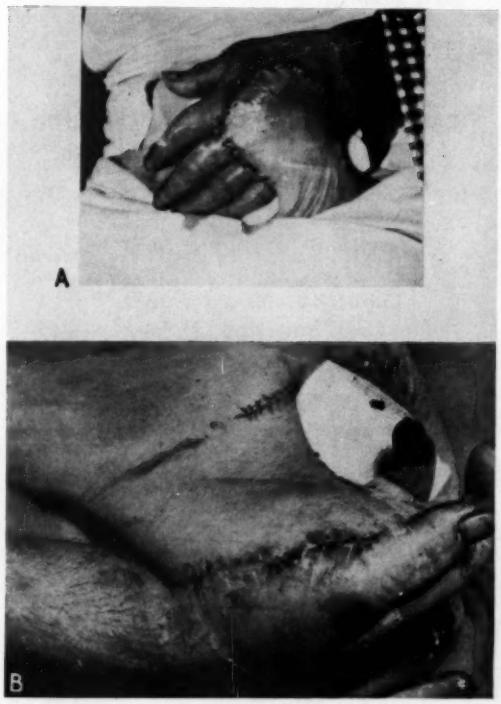

FIG. 6

(A) the epigastric flap is adaptable to dorsal wounds, the subcostal (B) to volar wounds.

important technical detail is to remember that the flap must be cut from the ipsilateral abdominal surface so that the hand

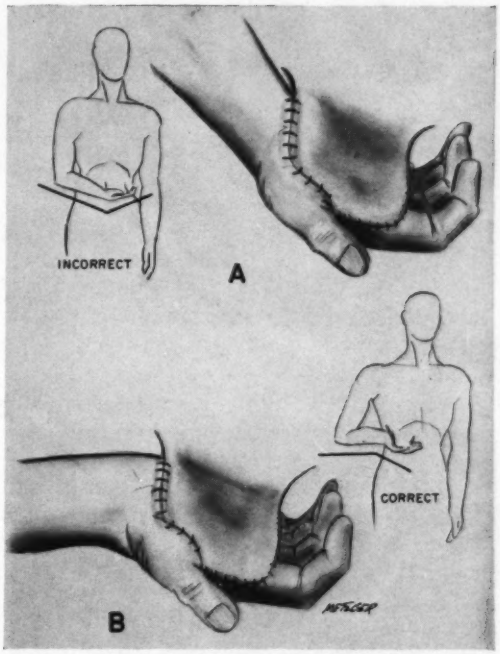

FIG. 7

Placement of the hand in the contralateral position (A) flexes the wrist and binds the shoulder and elbow. Placement in the ipsilateral position (B) maintains the normal balance and attitude while allowing for more liberal motion of the elbow and shoulder during the period of immobilization.

lies adjacent to the body rather than across it. The drawings in Fig. 7 illustrate this point, and the wrist flexion seen in Fig. 2 is a result of violation of this rule. This lateral position of the hand on the trunk allows normal moderate extension of the wrist and a far more liberal range of motion of the shoulder and elbow. In such cases there is usually no postoperative stiffness of these two important joints. To allow permanent fixation of shoulder, elbow, and wrist while resurfacing a hand is to violate the rule of proximal effectiveness for distal activity.

Following elevation of the abdominal pedicle and before attachment of the damaged hand, the abdominal defect and under-bridge of the flap are covered by a free split-thickness graft.[10] (Fig. 8, B) This ensures complete closure of all wounds and aids greatly in reducing infection, edema and fibrosis. Immobilization of these flaps can usually be effected with adhesive plaster instead of a cumbersome body cast.

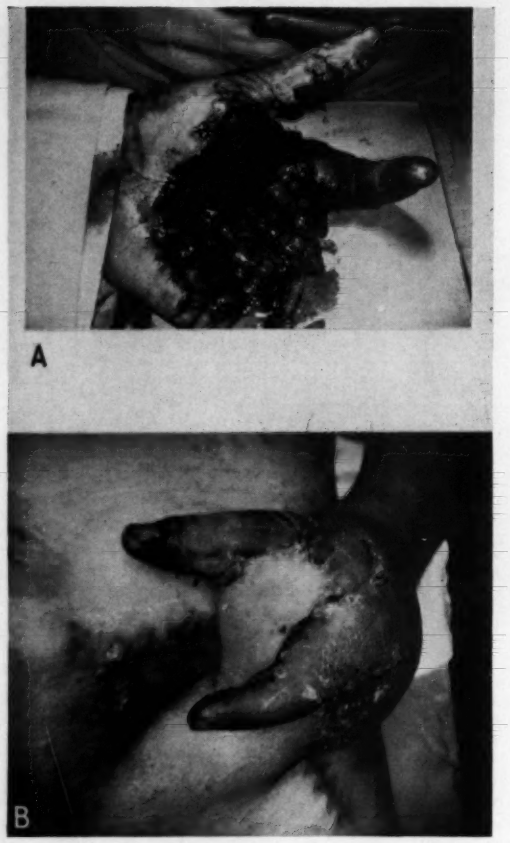

FIG. 8

(A) Blast wound of the hand requiring combined dorsal and ventral resurfacing with reconstruction of the thumb web. (B) illustrates the closure of the abdominal defect by a free split thickness skin graft.

The empiric time required for attachment of the flap is three weeks; however in severe blast injuries it is advisable to allow an additional ten to twelve days. The injured hand requires that long to lose its inflammation of injury and begin the attaching process. When division of the pedicle is contemplated a complete occlusion with a rubber-shod intestinal clamp should be a prerequisite. If no change in the circulation of the flap is noted, division can be safely carried out. Frequently division in stages (Fig. 6, A) can be done, so that the total length of time can be reduced by several days.

After division of the pedicle and adjustment of the flap to the wound, a snug pressure type dressing is applied and the hand is set again in the position of function.[5] A ten day postoperative period will usually allow full healing of the operative wounds, and a course of vigorous physical therapy can be started.

Not until maximum benefit from the immediate reconstruction has been attained can a revaluation of the hand for final reconstruction be made. These later procedures will, however, be lessened greatly by the early closure and meticulous care of the original injury.

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STRANGULATED DIAPHRAGMATIC HERNIA
A Report of a Case Complicated With Gangrene of the Stomach

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and
ALLSTON J. MORRIS, M.D.**
Wilmington, Del.

HISTORY

Ambrose Pare,[2] who lived between 1510 and 1590, was the first to give a detailed postmortem account of a strangulated diaphragmatic hernia. This he did in 1564. Occasional cases were reported in the early literature, but prior to the widespread employment of roentgenography the condition had seldom been correctly diagnosed before death. This condition remained more or less a medical curiosity for about 300 years. Bowditch, in 1853, collected 88 cases of diaphragmatic hernia available in the literature at that time and presented 1 case of his own. His own and one other were the only cases diagnosed before autopsy up to that time. Gibson, in 1930, reported 3 cases which he diagnosed clin-

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ically without aid of x-ray. The first case diagnosed by roentgen methods was in 1921, in the Mayo Clinic. In 1925 Morrison[8] suggested the Trendelenburg position to demonstrate a diaphragmatic defect radiologically. Since then the number of times this lesion has been recognized has increased steadily until now it is the second most frequent pathologic condition encountered in a large series of gastrointestinal cases, duodenal ulcer being the most common.

INCIDENCE OF DIAPHRAGMATIC HERNIA

In December, 1953, a mobile chest x-ray unit of the Delaware State-Wide Chest X-ray Survey visited the Delaware State Hospital and x-rayed patients and employees. Of a total of 961 patients, 7 were found to have diaphragmatic hernia (0.7%), according to Phillips.[10] It is estimated that slightly more than 50% of diaphragmatic hernias can be diagnosed in routine chest films. The probable incidence of diaphragmatic hernia in the over-all population is about 1%. Feldman[5] reported that the roentgenological incidence of hiatus hernia in 30,000 cases was approximately 1%. In 1,500 gastro-intestinal examinations, Schatzki[12] found an incidence of 3.5%. In 635 necropsies Eppinger[5] observed 11 cases, or 1.7%.

CLASSIFICATION

Diaphragmatic hernia has been classified by Astley Cooper into 3 categories: (1.) congenital, due to defects in the diaphragm arising from faulty embryologic development; (2.) acquired, which developed at points of anatomic weakness, that is, at the esophageal hiatus, aortic or caval openings; (3.) traumatic, that is, caused by rents in the diaphragm arising from direct or indirect trauma. Strangulation is infrequent in both congenital and acquired diaphragmatic hernias. Traumatic diaphragmatic hernias constitute over 90% of the cases complicated by strangulation.

INCIDENCE OF STRANGULATION

Carter and Guiseffi[2] compiled reports of 43 cases from 1798 to April, 1948. They included 4 of their own cases, 3 of which recovered and the other one died. The fatal case was that of a colored male patient, age 33, with a strangulated diaphragmatic hernia consisting of gangrene of the entire stomach and omentum. The correct diagnosis was missed completely and was made only at the operation. He expired shortly after surgical treatment. To these Pearson[9] later added 27 more from the literature and 4 of his own, to bring the total to 74 cases.

As far as gangrene of the stomach is concerned we have been able to find only 9 reports of gangrene of the stomach due to strangulated diaphragmatic hernia. Hamilton and Phillips in 1949 treated 2 cases of their own and collected 4 more from the literature. Since then, Pearson,[9] Hurley,[7] Hoffman et al.,[6] each treated a case.

Our presentation is the 10th reported case of strangulated diaphragmatic hernia with gangrene of the stomach. Two of these 10 patients survived operation.

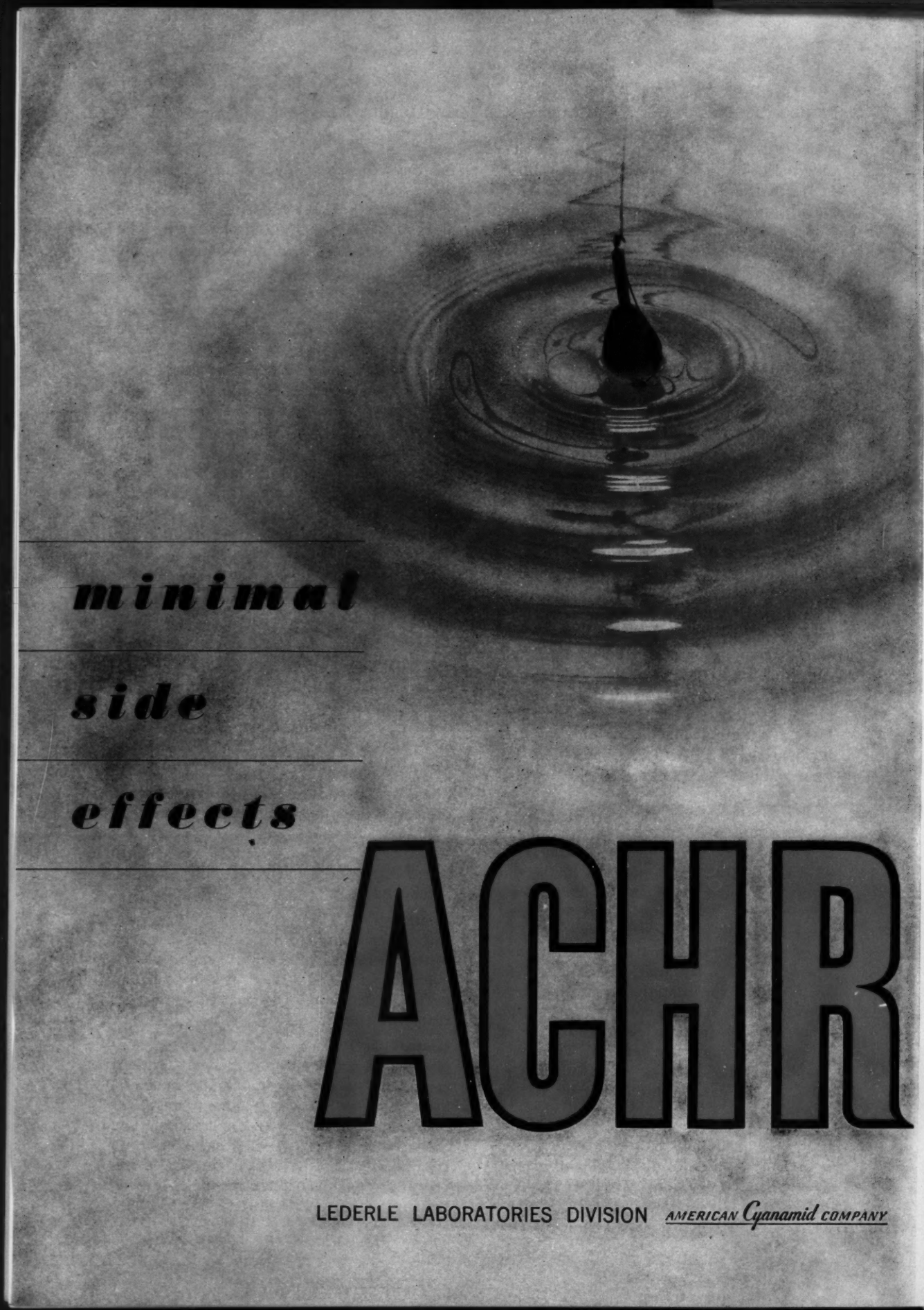
DIAGNOSIS OF STRANGULATION

Pain — Characteristically sudden, this has been the most usual initial symptom. The pain is in the epigastrium, upper left abdomen, or lower left chest. The pain may be moderate or severe. Left shoulder-top pain may be present.

Chest — There is diminished expansion of the left chest, and percussion yields dullness, or tymphany, of the left lower thorax. Adventitious sounds are usually heard. Dyspnea and dysphagia may be present.

Vomiting — This occurs in 88% of the cases, according to Carter. It is early, protracted and ineffectual, frequently with evidence of blood. It is usually concommitant with pain. Bile is usually absent from the vomitus.

History — There is usually a history suggestive of diaphragmatic hernia. Patient complains of epigastric pain or distress, heartburn or bloating, hemorrhage, or symptoms of acute intestinal obstruction. Since 90% of strangulated cases occur in traumatic cases, history of injury is important. There is often a record of a penetrating wound in the lower left chest.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X-ray — This is conclusive proof of hernia. A diaphragmatic hernia usually shows up on an ordinary PA chest film. Presence of free air in the hernial sac suggests gangrene of a viscus with perforation. The heart is usually shifted to the right.

CASE REPORT

C. V., Medical Record No. 217133, w.m., 62 years, was admitted to the Delaware Hospital August 10th, 1954, at 10 A.M. His complaint was that he ate something that did not agree with him. He stated that he was apparently asymptomatic until 10:30 P.M. of the previous day. His evening meal had been at 6 P.M. At 10:30 P.M. he had nausea and vomiting, which persisted throughout the night and the next morning. At the time of admission he was in semi-acute distress. His blood pressure was 214/104; TPR was 98.6°, 78, and 18, respectively. His chest showed increased resonance to percussion. The abdomen was soft and non-tender. Peristaltic sounds were audible. He had 5 previous operations (appendectomy, herniorrhaphy, prostatectomy, and 2 gall-bladder operations). His leucocyte count was 17,400. The differential count showed 81% segmented polymorphonuclear cells, 11% band polymorphonuclear cells, 6% lymphocytes, and 2% monocytes. Another count obtained later in the day showed 17.4 gms. of hemoglobin, 49% hematocrit, and 5,100 leucocytes. His differential count was 41% segment forms, 40% band forms of polymorphonuclear cells and 18% lymphocytes. The serum amylase was 128. His urine showed 2-plus albumin. At 5 P.M. a marked change took place in his condition. He was writhing in bed with pain in the epigastrium. He complained of pain in the left shoulder top. It hurt him to take a deep breath. On examination there was fullness in the epigastrium and there was board-like rigidity of both recti muscles. Peristalsis was diminished.

X-ray examination of the chest and abdomen showed a rather marked distension of the cardiac end of the stomach. There was elevation of the dome of the left diaphragm with free sub-diaphragmatic air. There was also a large hiatal hernia on the right side, containing a fluid level. Free air was noted in the hernial sac and the abdomen. (Figs. 1 and 2.) The radiologist felt that this represented a rupture of a hollow viscus, complicated by an esopha-

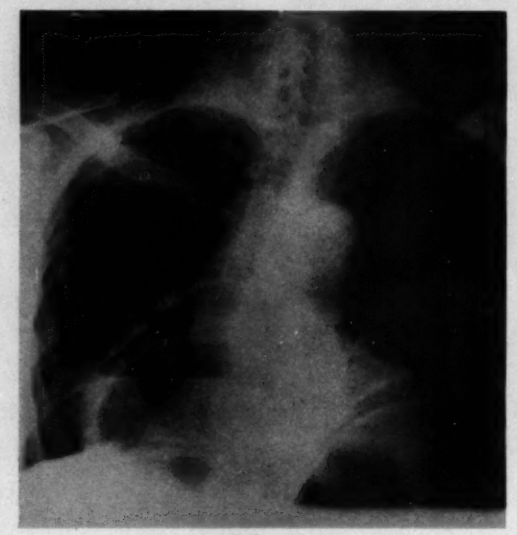

FIG. 1
Posterio-anterior x-ray view of chest and abdomen shows a large diaphragmatic hiatal hernia containing a viscus with a fluid level. Free air is noted in the hernia sac and beneath the left diaphragm.

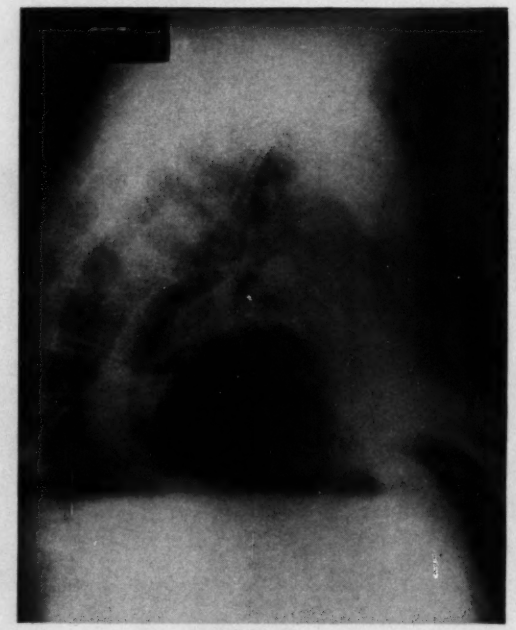

FIG. 2
Lateral x-ray view shows free air under diaphragm and in the upper anterior peritoneal cavity.

geal hiatal hernia on the right side. The patient was taken to the operating room about 10 P.M. Under endotracheal anesthesia the abdomen was opened. A large mass of omentum was removed from the esophageal hiatal hernia. The whole hand could be inserted into the hernial sac, extending 4 or 5 inches above the diaphragm. The hernia extended into the mediastinum and into both left and right chest. The upper fundus was not visualized because of the high dome of the left diaphragm. However, it was carefully palpated and no perforation was found. The rest of the stomach was examined anteriorly and posteriorly for perforation, but none was found. The rest of the G. I. tract was carefully examined and no perforation was noted. It was then decided to open the left chest, which was done by removing the 8th rib. The esophagus was examined and found to be intact. On opening the dome of the diaphragm the top of the gastric fundus was gangrenous and had a perforation 2 cm. in diameter. The entire gangrenous area measured approximately 6 cm. in diameter. Wedged into this area there was a handful of undigested spinach and corn, which accounted for the negative findings during the abdominal exploration. The gangrenous area was excised and closure of the stomach was effected through healthy gastric tissue. The diaphragm was closed and the hernia was repaired. The patient's condition was not good. During the course of the operation, he had received 4000 cc. of blood.

His condition remained fair after the operation. His blood pressure had to be maintained with the use of norepinephrine. Oxygen was given continuously by mask. He expired on August 12th at 6:50 A.M.

Autopsy was performed. The resected stomach was intact. There was no evidence of any peritonitis. The lungs showed marked congestion. The hernial repair was satisfactory. The final diagnosis was: (1.) shock, post-operative; (2.) strangulated diaphragmatic hernia, with gangrene of the stomach.

COMMENT

It is amazing that the patient did not have more acute symptoms initially, but this is borne out by other cases in the literature. Pearson reports a fatal case of gangrene of the entire stomach with few symptoms and physical findings.[9] Carter and Guiseffi also presented a similar case with mild upper left abdominal tenderness.

This patient had a perforation of the cardiac end of the stomach due to gangrene, without any history of preceding trauma. We have assumed that gangrene was secondary to strangulation of the stomach in the mediastinal hernial sac, although at operation the stomach was found free in the abdomen, and the opening into the hernial sac was large enough to admit the surgeon's hand. Our assumption is based on the following points: (1) the presence of a long fluid level in the chest, which seems almost necessarily to have been in an organ as big as the stomach; (2) Cohen has stated that "experimental data . . . seems to indicate that if infarction of the stomach is to be produced, both arterial ligation and venous impediment are requisite".[3] At autopsy, no evidence of local vascular disease was present except for that clearly associated with the acute episode. Therefore, it is felt that some severe insult such as strangulation is necessary to explain the gangrene.

This case of gangrene of the stomach due to a strangulated diaphragmatic hernia, is indeed a rare condition. The fact that the lesion could not be identified through the abdominal incision was also an oddity. Eventration of the left diaphragm, no doubt, helped the difficulty.

With this case in mind, and our previous experience, we propose a plan for handling diaphragmatic hernias.

1. Repair all cases when first diagnosed in patients under 50 years of age, in view of the fact that they usually cause difficulty later merely by their increasing in size.

2. Repair the hernias having complication in patients over 50 years of age. These complications may be any one of the

following: pain, hemorrhage, ulceration, strangulation, dysphagia, and dyspepsia. Very frequently the first "attack" is diagnosed as a coronary occlusion and the patient is known as a cardiac for the rest of his natural life.

Most mild symptoms will be relieved by an ulcer regime, semi-Fowlers position, or a left phrenic crush. A phreniclasia, if properly performed, in the elderly patient, will produce a dramatic and gratifying result.

The surgical repair should be aimed at re-establishing a normal "pinch-cock" at the distal end of the esophagus by (a) preserving good muscle fibers of the right diaphragmatic crus, (b) producing a normal subdiaphragmatic angulation, and (c) re-approximating the esophageal gastric ligaments.[4] The ease of repair makes the trans-thoracic approach most ideal for operations on diaphragmatic hernia.

SUMMARY

A case of strangulated diaphragmatic hernia with gangrene of the stomach and perforation is presented.

Historical background, classification, incidence and symptomatology for diagnosis of strangulated diaphragmatic hernia are discussed.

Immediate surgery and resection of the stomach are essential in the successful treatment of a strangulated diaphragmatic hernia complicated by gangrene of the stomach.

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CLINICOPATHOLOGIC CASE REPORT

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RAY G. SARVER, M.D.,[b]
and
RICHARD J. COLFER, M.D.[c]
Wilmington, Del.

PRESENTATION OF CASE*

Dr. Vandervort. This 25 year old married negress was admitted to this hospital on April 27, 1954. The patient was comatose and all available history was obtained from her husband.

The patient had been in her usual excellent state of health until six weeks prior to admission when she began to complain of severe headaches and dizzy spells. Three weeks prior to admission these complaints were of such magnitude that the patient no longer was able to maintain her housework satisfactorily. No fever was noted. No personality changes nor localizing neurological symptoms developed. Vague abdominal pains were present for three weeks prior to admission. On April 25 the patient was seen in the emergency room at her local hospital with the complaint of severe headache and abdominal pain. These pains were eased with demerol, but within a day the patient returned in a comatose state. Following the administration of an unknown medication the patient appeared to be improved and was ambulatory but she soon became comatose again and was referred to this hospital.

There were no previous operations, hospitalizations, or serious illnesses. The patient was grava I, para I. The one pregnancy resulted in an uneventful delivery seven months prior to the onset of the present illness. During this pregnancy the CBC, serological tests for syphilis and blood pressure, were supposedly normal. Postpartum course was uneventful except the patient had had no menstrual period since pregnancy.

Laboratory studies at outside hospital: WBC. 10,400, with 56 polymorphonuclears, 4 non-segmented polymorphonuclears, 40 lymphocytes; RBC. 3.2 with Hb. 63%; 2 plus albuminuria; 2-3 WBC's; few granular casts; BUN. 9 mg%; blood sugar 50

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* Delaware Hospital Case No. 213601 presented at Staff Clinical Pathology Conference.

mg%; CSF: 3 cells, sugar 47 mg%, protein 35 mg%.

On admission at this hospital the patient was comatose and non-responsive to all stimuli. She was thin and appeared acutely and severely ill. BP 70/40, P 140, R 36, T 106 F.

No rash, cyanosis or icterus was present. The left tympanic membrane was slightly red. The mouth was held tightly closed. The lungs were clear after bronchial secretions were aspirated. There were no murmurs. The heart was not enlarged. A tachycardia of 140 was present. The abdomen was soft, with no organs or masses palpable. Pelvic and rectal not remarkable. The fundi revealed normal discs, venous engorgement and tortuosity. Neurological examination disclosed hypoactive reflexes with no pathological reflexes. The corneal reflex was absent. The pupils reacted poorly if at all to light.

A questionable traumatic spinal tap revealed clear fluid with 9 WBC's and many RBC's, with initial pressure 80 mm. Hg. The Hb was 13 grams. WBC 8,400 with 44 polymorphonuclears, 20 non-segmented polymorphonuclears, 32 lymphocytes, 2 monos, 2 eos. The urine had a sp. gr. of 1015, 2 plus albuminuria, 4 plus glucosuria (with 10% travert running intravenously). A portable chest x-ray was within normal limits except the cardiac shadow appeared to be rather small. A ventriculogram showed some air in both ventricles. Some irregularity of the left lateral ventricle was noted which was attributed to incomplete filling. EKG demonstrated nonspecific lowering of T waves. EEG could not be read because movement resulted in constant artifacts. Urine, CSF and blood cultures collected on admission failed to grow any organisms. The blood cultures were incubated for 15 days anaerobically and aerobically. Febrile agglutinins were negative. (Typhoid - negative; para-typhoid A&B - negative; Brucella - negative; Proteus OX19 - negative). Blood drawn and sent to the Virus Diagnostic Lab was negative for lymphocytic choreomeningitis, mumps, Eastern, Western, St. Louis encephalitis. Various other chemical and electrolytic studies were performed and their values may be determined from the Tables. PPD#1 and Serological tests for syphilis were both negative.

On admission, aqueous penicillin 1,000,000 units was given, q. 2 h I.M. plus streptomycin 1 gram q. 12 h I.M. Intravenous fluids were given daily to maintain a fluid intake of 2-3 liters daily and an output of 1-2-.5 liters. The temperature spiked between 102-103 at first and then 101-102. Sponges were applied to keep the temperature below 103. On May 3, six days after admission, the antibiotic was changed to procaine penicillin 300,000 units daily. The blood pressure averaged about 90/60 the first three days, requiring levophed initially to maintain it at this level. The last few days the pressure remained about 100/70 without levophed. The patient remained comatose. A tracheotomy was done May 3 for respiratory distress secondary to tracheal secretions. She expired May 4, seven days following admission.

SUMMARY OF LAB. STUDIES

4/27	4/28	4/29	4/30
Hbg 13	13.5	10.6	Platelets 193,000
RBC	3.9	3.2	Total eos 69 mm
WBC 8,400	21,200	11,900	L E test negative
Polymorphonuclears 44	18	23	Serological tests for syphilis - negative.
Non-segmented polymorphonuclears 20	45	44	
Lymphocytes 32	31	24	
monos 2	5	8	

Feasting sugar	234		
Fasting sugar		164	88 86 101
BUN		12mg	
CO_2 17mEq		17mEq	
Na		17mEq	138mEq
Total protein with a/g	5.7 with 3.2/2.5		
Bilirubin	.39 direct; 1.2 indirect		
Methyl alcohol test	Negative		

Urinalysis	4/26	4/27
sp. gr	1015	1016
Albumin	2 plus	2 plus
sugar	4 plus	Negative
RBC	1-4	10-15
WBC	2-3	0

few fine, hyaline casts.

Urine negative for arsenic, mercury and barbiturates on 4/28.

DIFFERENTIAL DIAGNOSIS

Dr. Sarver. The history of the illness in this case is that of a process which seems to have involved the central nervous system of a healthy young adult and was fatal in a short time, yet there was never much in the way of physical signs or laboratory findings to localize it. It is interesting to speculate where in the brain such a lethal but obscured lesion could occur and what produced it.

Six weeks prior to her admission this 25 year old negro woman began to have severe headaches and dizzy spells which worsened to the point that she could not do her housework. There was no fever, personality change, or focal neurological symptom noted. The physician who saw her on April 25 apparently found no physical signs but within a day the patient returned in a comatose state. Her apparent improvement after medication suggests strongly the possibility of a hypoglycemic reaction. However, the blood sugar reported as 50 mgm % was never confirmed later, and can be further questioned because the CSF sugar, which is roughly one-half the blood sugar in the absence of CNS infection, was normal. The other laboratory findings are not remarkable. We would like to know if she had a temperature or was given antibiotics at this time.

The past medical history confirms the fact that she was in good health before the present illness. Seven months ago she had given birth to a child without any complications, and yet she had had no menstrual periods since then. Amenorrhea of this duration is physiologic at times during lactation and it would be of interest to know if she was nursing her baby. She could be pregnant again; however physical findings did not substantiate the fact. Cases of the Chiari-Fromel syndrome are known in which there is persistent postpartum lactation and amenorrhea occurring usually in primiparous women and resistant to all therapy. Finally, we must consider endocrine disturbances, particularly pituitary insufficiency as a cause of her amenorrhea.

When seen at the Delaware Hospital for the first time the patient was in coma, with a high temperature and shock. The physical examination gave very little in the way of localizing signs. Absence of the corneal reflexes and sluggish pupillary reflexes could mean brain stem involvement or merely severe CNS depression from any cause. Whether the fact that the jaw was closed tightly has importance is again hard to say. Perhaps the lack of neurological findings is in itself the most striking thing. A temperature of this magnitude certainly suggests sepsis, some hypothalamic lesion, or massive cerebrovascular accident.

It is necessary then to consider the causes of coma, and we can weigh their possibility with the facts at hand as we go. Coma can occur as the terminal stage of any illness, but it is the case with acute coma which offers most difficulty in diagnosis.

First of all are the group of conditions in which the brain is directly involved. Head injury is an important one, but in this case we have no history or physical findings to suggest it. Subdural hematoma can occur acutely following head injury or vascular accidents, but the findings on spinal and ventricular tap make it unlikely. Epidural hematoma is likewise associated with injury, often followed by a lucid interval then the development of hemiparesis, a dilated pupil, and coma.

Cerebral hemorrhage can occur in the cerebrum, the cerebellum, the ventricles, or the brain stem. As a rule the symptoms are dizziness, headache, and confusion or unconsciousness, stertorous respirations, a rapid full pulse and the temperature is subnormal or elevated to 101 or 102 degrees F. The more common causes of cerebral hemorrhage in young persons are syphilis, hypertension from any cause including coarctation of aorta, mycotic aneurysms, t r a u m a , blood dyscrasias, brain stem tumor, chemicals, angiomas, and not rarely there is no apparent reason. The cardinal point to be remembered is that even during complete unconsciousness neurologic localization is seen.

Cerebral embolism occurs in the younger age group, mostly in persons with auricu-

lar fibrillation and subacute bacterial endocarditis. To diagnose it one must have a primary site, which was not present in this case.

Subarachnoid hemorrhage is a relatively common type of vascular accident in young persons. Usually severe headache is present, a stiff neck develops, and the spinal fluid contains red blood cells. Signs of hemiplegia are not present as a rule, but meningeal signs and bloody spinal fluid are so prevalent in these cases that our patient does not seem to fit the picture. The RBC found on the second tap are not reported as crenated and the fluid was not xanthochromic, so the tap was probably traumatic. The other possibility is an aneurysm rupturing into the brain stem, but here again localization is very constant, just as in cerebral hemorrhage. Our patient held her jaw tightly, but presented no other localizing findings, and this single finding is hard to evaluate.

Encephalitis as a cause of coma often begins with fever, following which obvious evidence of brain involvement occurs and this is often in the brain stem giving ocular palsies, peripheral facial weakness or difficulty in swallowing. Here we at least have the negative virus studies plus the lack of localization to make diagnosis of encephalitis precarious.

Meningitis must be considered, but can be ruled out on the basis of the CSF findings and lack of meningeal irritation. On the other hand, a brain abscess can occur anywhere in the brain and the CSF is quite often normal in cell count, and sterile on culture. In making this diagnosis a history of a site of infection is as a rule given much weight.

If this were a brain tumor we should expect an increase in CSF pressure, findings on ventriculogram, and focal signs of some sort.

Syphilis of the CNS can mimic any syndrome and must always be considered. We have a normal serology reported during her pregnancy and a repeat serology at the Delaware Hospital was negative.

Therefore, of the causes of acute coma involving the brain directly, it is hardest to eliminate brain abscess, aneurysm rupturing into the brain stem, and encephalitis with an unusual localization in the brain stem.

The other category to be considered are diseases not primarily involving the brain. Severe fever from any cause produces coma, and is seen in cerebral hemorrhage, midbrain tumors or hemorrhage involving the heat regulatory centers of the hypothalamus, heat stroke, and severe infections, particularly miliary tuberculosis and meningococcus infections. The patient we are considering had fever and leucocytosis with a shift to the left, but all cultures and agglutination tests were negative. The PPD was also negative but this is not rare in terminal miliary tuberculosis. Nevertheless, we have to remember that the patient probably got antibiotics as soon as she became febrile and before cultures were drawn. Also, she was severely ill six weeks before there were any signs of infection.

Shock due to massive hemorrhage from the lung, intestine, ectopic gestation, or aortic aneurysm should be ruled out, but our case from history, physical examination, or lab findings, does not fit any of these.

Thought must be given to the group of metabolic diseases including diabetes mellitus, hypoglycemia, Addison's disease, myxedema, and Simmonds disease. Diabetes and hypoglycemia are ruled out by the laboratory findings. Addisonian crisis is an interesting possibility and usually presents with symptoms of previous lethargy, severe abdominal and leg pains, vomiting and epigastric discomfort, cold extremities, subnormal temperature, low blood pressure, scanty urine, and often a rigid abdomen. The inconsistencies in our case are hyperthermia and no previous trouble or incident likely to produce a crisis. Also, we would expect hemoconcentration, increased plasma proteins, increased hemoglobin and hematocrit, low serum sodium, increasing BUN, and elevation of the eosinophil count. A serum potassium determination would have been useful in ruling out Addisonian crisis. Before dismissing this diagnosis, it is well to remember that bilateral adrenal hem-

orrhage can only be diagnosed at postmortem, and one should treat all patients with signs of vascular collapse and poor response to chemotherapy as potential cases of acute adrenal insufficiency. Especially is this true in stress and overwhelming infection.

In pan-hypopituitarism amenorrhea is an early and constant occurrence and the old concept of extreme cachexia is the exception, not the rule. Decrease in thyroid and adrenal function are often not complete and present a mixed and confusing picture. Simmond's described a disease which follows partuition and the patient deteriorates rapidly. His first case occurred in a woman with puerperal sepsis and he interpreted his findings as being the result of a bacterial embolus. Today the consensus is that thrombosis is the more frequent cause of hypophyseal necrosis and one common factor is postpartum shock. Endocrinologists today feel that pituitary necrosis occurs more often than was formerly recognized, minor degrees being detectable only by newer biochemical methods, and not being fatal. Certainly it is one place in the brain which is silent neurologically and yet a lesion here could produce rapidly fatal illness. For that reason it remains a good possibility in this case.

Uremia produces coma but is usually easy to diagnose because of hypertension, dehydration, vascular retinitis, increasing BUN, low CO_2, a fixed urine specific gravity of 1010, massive albuminuria, and casts.

Cardiac arrythmias are also capable of producing syncope and coma but were not present in this woman. Finally, massive liver disease often ends with coma, but this patient has a normal bilirubin, no ascites, and normal serum protein.

Many exogenous causes of coma are known: lack of oxygen, lack of nicotinic acid, alcohol and various intoxicants. In intoxication the history is of paramount importance. Often there are no localizing signs, abnormal urine, blood or CFS findings. It should be considered especially when coma is afebrile.

In most cases the toxin produces a characteristic clinical pattern. For instance, opiates depress respiration and constrict the pupils. Barbiturates depress the entire central nervous system with progressive loss of reflexes and respiratory depression, and can, in questionable cases, be found by special tests of the urine. Characteristically the temperature is decreased. Bromide is a CNS depressant with little effect on most other tissues and is at times accompanied by an acneform rash. Phenol affects the m e d u l l a and causes early convulsions. Methyl alcohol causes an odor on the breath, rapid respirations, and dilated pupils, and is detected by chemical means. Benzene and carbon tetrachloride cause severe gastrointestinal irritation when ingested, plus other specific toxic effects. Cyanide poisoning is extremely rapid and kills by asphyxia and there is a bitter almond smell to the breath. Carbon monoxide poisoning is not a diagnostic problem unless the history is totally lacking. Methemoglobinemia due to nitrate or chlorate ingestion produces intense cyanosis and a typical chocolate color to the blood. Mercury affects primarily the kidneys and kills by renal failure. Arsenic ingestion produces a severe gastroenteritis with persistent vomiting and diarrhea. Neither the clinical or laboratory findings in this case suggest intoxication.

In summary, it seems that the patient was afflicted by a process which first involved the CNS without producing much in the way of neurologic localization, and yet the process was fatal in a short time. At first there was no suggestion of an infectious process but even though cultures were always negative the high fever in the terminal stages suggest sepsis. A hypothalamic lesion, either hemorrhage, encephalitis or tumor, could have caused the high fever but it is hard to picture such a process without any o t h e r neurologic changes. The anterior pituitary, if destroyed partly by postpartum necrosis and later formation of an abscess would be silent neurologically and yet capable of causing the patient's clinical picture and

demise. The most likely diagnostic possibilities might be listed as follows:

CLINICAL DIAGNOSIS

1. Pituitary Lesion
   a. Abscess
   b. Necrosis
2. Hypothalamic destruction due to
   a. Ruptured arterial aneurysm
   b. Encephalitis

AUTOPSY FINDINGS

Dr. Colfer. The cerebral vessels appeared normal. No evidence of abscess, tumor or hemorrhage were noted on serial section. The brain tissue appeared wet. Examination of the pituitary gland revealed abnormal consistency suggesting sepsis. Microscopic sections of pituitary showed necrosis, edema, and acute and chronic inflammation. A postmortem culture from the pituitary gland revealed a hemolytic enterococcus resistant to penicillin and tetracycline, and a hemolytic staphlococcus aureus, coagulase positive, resistant to penicillin and tetracycline.

PATHOLOGIC DIAGNOSIS

Pituitary necrosis

Cerebral edema

SOME COMMON SENSE CONCEPTS IN THE MANAGEMENT OF ACNE VULGARIS

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Our experience with the observation of over five thousand patients afflicted with acne vulgaris has led us to certain therapeutic concepts. While there are many theories concerning the etiologic aspects of acne, it is an accepted fact that the pathogenesis of this most common dermatologic problem is an overactivity of the sebaceous glands resulting from pilosebaceous stimulation. Unfortunately, nature has endowed us with a preponderance of sebaceous glands on the face, chest and back. The reason for this is quite obvious, as these sites lack subcutaneous fat and the sebaceous glands act as an insulating medium.

Although acne vulgaris is primarily a teen-age problem, it may extend long beyond this period, or may occur initially after the second decade in life. While we are concerned most often with the problems of the teen-age group, the basic factors in management apply also to older groups of patients.

From the onset it is important to inform the acne patient that the therapy does not propose to cure their acne but attempts to clear their blemishes, and advice is given to them as to the manner of keeping their acne under control until they shed their adolescence.

A prime consideration of the therapeutic approach is to attempt to prevent the most serious sequela—the resultant scarring. It is well-known that acne is a scarring disease. In the dim past it was commonly referred to as "stone pox." Proper treatment, instituted early, will prevent this to a great degree. Neglect may result in mild pitting to severe facial disfigurement, with the sequela of dire psychic trauma.

FUNDAMENTALS OF THE MANAGEMENT

The ideal treatment begins by educating parents and the patient of the need of proper care of the skin, the early recognition of acne, early institution of proper treatment, and the importance of controlling this disease. Unfortunately, acne is one of the most commonly neglected skin diseases because of the average parent's attitude that time will usually clear up the blemishes. The word "time" usually includes puberty through adolescence and marriage, including the birth of children, all of which means that nothing specific is done for the patient.

ENDOCRINE FACTOR

We do not employ any endocrine therapeutic methods, despite the endocrine relationship of acne vulgaris. Our results,

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using this therapeutic measure, have been disappointing. Although medical literature abounds with glowing reports, there are also a just number of inconclusive and contradictory findings which do not warrant the employment of sex hormones.

Thyroid preparations are indicated only when there is definite clinical evidence to substantiate laboratory findings of thyroid deficiency. This drug should not be employed indiscriminately.

DIETARY FACTORS

The role of dietary indiscretion has been over-emphasized in the treatment of acne vulgaris. Our experience has led us to believe that chocolate is the only incriminating food and it is routinely insisted that all acne patients abstain from chocolate in every form. All too frequently have we observed underweight patients placed on a rigid diet only to lose more weight, wherein they are apt to have an exacerbation of their acne. Generally speaking, if patients are underweight attention is directed toward building them up and improvement of their general state of health. If they are overweight a rigid diet may be invoked. Iodides, in the form of iodized salt and seafood rich in iodine are contraindicated, since iodides produce an exacerbation of acne.

VITAMINS

Vitamin-A purportedly exerts a beneficial effect on the pilosebaceous system; however, the results obtained in the treatment of acne are inconclusive. Vitamin-A should be given orally in dosage of 50 to 100,000 units daily during the fall and winter months. Synthetic vitamin-A, (Vidom-A) is a satisfactory preparation. Large doses of Vitamin C have been advocated.

Multiple vitamin therapy exerts a beneficial effect and is indicated in patients below par. Often teen-agers suffer from a secondary anemia. Ferrous sulfate may be incorporated with the vitamin therapy if need be, as a supportive measure.

CONTROL OF SEBACEOUS ACTIVITY

Keeping the skin dry, thereby controlling the sebaceous activity, is the basic local therapeutic measure. This can be accomplished chiefly by physical and local measures.

The most important physical modality at our disposal is the x-ray. In experienced hands fractional doses of x-ray may be instituted in patients sixteen and over. X-ray therapy does not produce scarring. However, I firmly reiterate, it should be employed only by physicians who have been properly trained in this technique.

Local measures, and detailed instructions as to their use, as well as insistance on their use with cooperation from the patient, go a long way in the treatment of acne. Some consider the topical management of acne more important than the systematic approach, and the topical measures required vary as widely as in other dermatologic conditions. The vast majority of patients with acne have excessively oily skin and, therefore, the production of slight dryness, and subsequent desquamation, is desirable. This helps to keep open the sebaceous orifices and discourages comedone formation. Creams, ointments and similar oleoaginous vehicles are generally undesirable. "There are innumerable proprietary remedies for acne which are often unnecessarily complicated and may contain ingredients which will give rise to irritation and sensitization of the skin."[1] The very multiplicity of these products demonstrates inherent failings of each, and consequently these are but short-lived.

Each method or remedy for the local therapeutic approach to acne has its champion among the medical and pharmaceutical professions. However, in a disease whose manifestations may range from the mild to the severe, it is necessary to have a flexible plan which is capable of being adapted to the individual patient needs. It is important to emphasize that at present there is no cure for acne. Symptomatic relief and temporary remission of the disease are the present

day goals and achievements until abnormal pilosebaceous activity subsides. No single form of therapy is all-inclusive.

Sulfur therapy has certain clinical advantages, and is used chiefly for its mild keratolytic, astringent and drying properties. It serves as a valuable therapeutic agent in the mild cases, and as an adjunct to other therapy in the severe cases. The use of topical preparations in conjunction with other well-known regimens affords the best approach available to date.

A new topical sulfur preparation was recently introduced and which has been found to be very effective. This preparation, Pronac, consists of a stabilized sulfurated potash coated with an equimolecular film of zinc sulfate separated from each other by a layer of an inert colloid. When Pronac is added to water the instantaneous solution of zinc sulfate occurs. The aqueous dispersion of the colloid permits the solution of the sulfurated potash, thereby causing the reaction to take place. In addition, the inert colloid serves to suspend the precipitated materials and thereby prevents their clumping and promotes a firmer adherence of the solid particles to the surface of the skin. The colloid also acts to retain the gases evolved through colloidal adsorption, thereby reducing any pronounced sulfuraceous odor.

This product is packaged in an individual dosage container, for the patient to mix before using, thereby having available a reproducible lotion containing all the therapeutically active ingredients of the freshly prepared product each time.

The patients were instructed to apply the freshly prepared lotion at night and to thoroughly cleanse the skin in the morning. The results obtained with this preparation were most gratifying and were evident within a minimal period of time. There was a high order of astringency and drying-action observed after the use of this preparation. When applied to the skin the preparation did not flake off and the close contact of the drug with the surface permitted a more efficient activity.

Some patients may require a lesser dosage of the sulfur-compound, while others may need a high concentration. A blond, fair-skinned patient will usually require a lesser dosage than will the brunette. The flexibility afforded is a particular advantage of the patient-prepared lotion because dosage may be varied to the needs of the patient.

Occasionally a patient may be found to be sensitive to sulfur, producing a local reaction. Then it is necessary to change to a simple lotion employing mercury as an antiseptic, bearing in mind that some patients may also be mercury sensitive. The following prescription may also be prepared without mercury if necessary.

Rx:	Hydrarg. Chloridi. Coros.	0.1
	Aqua Hamamelidis	60.0
	Isopropyl Alcohol 70%	60.0
	Aqua rosia q.s.	180.0
Sig:	Apply locally to affected area three times daily and nightly.	

Greases, such as cold cream, pancake make-up, and greasy foundation lotions, are contraindicated in patients who have acne. However, as the lotions begin to dry the skin, a bland, neutral ointment may be used at night in conjunction with the lotion. Acnophil ointment has served my patients very well in this regard. If the skin is still too dry and appears irritated, stop the lotion for a few days and apply a bland ointment.

The choice of soap is important. If the skin is very oily, especially in the summertime, a soap that will have a high defatting action is indicated — Lava, or preferably a laundry soap, such as Fels Naphtha or yellow Octagon. This may sound harsh to the patient but it will help considerably. If the skin should become too dry change to the milder toilet soaps available on the market.

Astringents are very beneficial, especially after washing the face before bedtime. The skin should be properly cleansed before applying the nightly application. Sebaclen, an acetone preparation, is an excellent one. Destina is a milder but very pleasant astringent.

Auxilliary Precautions

Concomitant seborrhea should always be treated. With the advent of the medicated shampoos this is a simple measure. Selsun suspension is a good preparation. Weekly shampoos are recommended. Teles suspension is another excellent preparation and is much more pleasant to use since the patient does not have to use a soap before and after the special shampoo. It is urgent that diseased tonsils and abscessed teeth be removed. I have observed many recalcitrant pustular acne lesions subside after the removal of such foci.

Oral and Parenteral Therapy

In very recalcitrant deep cystic pustular acne lesions and in acne conglobata, sulfonamides, antibiotics, and foreign protein therapy are indicated until there is a visible subsidence of the lesions. These preparations should never be employed routinely in the average acne patient because of danger of inducing a sensitivity. Terramycin tablets, 250 milligrams, four times a day for one week, then twice a day for two weeks, is a satisfactory regimen, bearing in mind that vitamin therapy should be prescribed concurrently with this antibiotic therapy.

In very resistant acne lesions 2 cc of lacto-protein may be administered intragluteally weekly for a period of six to eight weeks, depending on the response.

Emotional Factors

Acne and its unfortunate sequela of scarring always produces a psychic trauma. Patients need reassurance, moral support, understanding, and patient parents, and above all mild sedation to traverse this crucial period. Bromides are definitely contraindicated since they produce an exacerbation of the acne lesions and are conducive to the production of pustular lesions. There is usually a flare-up of the acne lesions during periods of stress such as school examinations, etc. We must bear in mind that the teen-age is usually a stage of emotional instability and a keen understanding of this group of patients and their problems is essential.

Local Office Procedures

I do not believe in removing comedones. Not only are you unpopular with your patient but sometimes pustulation follows. The patient feels that if a doctor can pick, he or she has an incentive to pick. The most important commandment in treating acne patients is "Do Not Pick." Keratolytics and physical measures such as ultraviolet and cold quartz irradiation will produce an exfoliation which will eventually take care of the comedones. Milia may be removed by pricking the top of the lesion and expressing the contents with a comedo extractor.

Some acne surgery may be indicated. Sebaceous cysts may be removed with the minimum of scarring by penetrating the cysts with a sharpened applicator dipped in phenol, and expressing the contents through a small opening. The sac is not expressed and may then be destroyed by applying phenol in situ, or blanching the lesion with monopolar electro-desiccation; the sac will then separate and may be expressed simply in a week or ten days following the procedure.

Local chemotherapeutic measures are very beneficial and desirable to produce an exfoliation, mild to severe, depending upon the indication. First cleanse the skin with acetone or a suitable astringent, then a mild keratolytic may be painted with an applicator on all of the affected areas. Then apply a cosmetic lotion base to neutralize the keratolytic. This procedure may be repeated as often as necessary at the discretion of the physician.

Care of the Skin

A mimeographed sheet with the detailed instructions on the care of the skin is important to the acne patient. However, it must be remembered that this is to be modified to suit the individual patient, particularly the dietary measures and addition of other therapeutic measures to take care of their skin properly may be necessary.

INSTRUCTIONS FOR PATIENTS WITH ACNE VULGARIS

1. Wash the face gently, (avoiding massage, missing the sorest places) with plain soap or a stronger soap if the skin is oily. The face should be washed at least three times a day.
2. Apply the prescription with the fingers over the entire area of oiliness and eruption, working it gently into the skin as directed.
3. No grease of any kind is allowed on the face unless especially prescribed. Rouge (not cream rouge) powder and lipstick are all right.
4. Keep the fingers off the face. Keep hair away from the face at night.
5. Shampoo weekly or as directed, with special prescription shampoo.
6. Get plenty of sleep and rest, at least eight hours each night.
7. Do not exercise violently. Perspiring is harmful as it stimulates the oiliness of the skin. If you should perspire freely use the astringent which was suggested, as often as possible.
8. Avoid sunburn. An excessive amount of sun stimulates the skin and produces more oiliness. Before exposure to the sun use a protective preparation, such as Raynox or Skolex. If either one of these preparations is applied it will produce a tan and will serve to filter out the harmful rays of the sun. There is usually an aggravation in November, December, and January, and people cannot understand why their faces are worse at that time. The reason for that is the previous summer sun.
9. Maintain regular bowel habits; learn to live without laxatives. Allow time in the morning to have a movement; do not procrastinate when the urge comes on; disregard a missed day, for the bowel content is not poisonous.
10. Avoid rich cream, homogenized milk, mayonnaise, chocolate, cocoa, soda fountain drinks, alcoholic drinks, nuts, peanut butter, thick gravy, greasy and fatty foods, excessive amounts of tea and coffee. Eat moderately of ice cream, butter, cheese, bread, pastries, and starch foods generally.
11. You may have bread, potatoes, eggs, meat of any kind, except pork and sausage, all vegetables, all fruits, fish, seafoods except those rich in iodines, such as shrimp, oysters, etc.
12. Do not use iodines in the form of iodized salt, or ask your family physician if he is prescribing iodides in the event that you are under treatment for acne. Do not use bromides in the form of bromo-seltzer, bromoquinine, or ask your physician if he is prescribing bromides.
13. Do not pick your face if you wish to avoid scars. This is most important. Do not expect a cure in a few treatments. It takes time. Follow instructions, and disregard any advice your friends might give you in reference to your skin trouble. It will only be confusing.

TREATMENT OF ACNE SCARS

There are three general measures available in the attempt to improve scarring resulting from acne: (1) cryotherapy; (2) local chemotherapy; (3) abrasive mechanotherapy. *Cryotherapy* consists of the local application of solid carbon dioxide (or dry ice) in the solid form, or in combination with acetone in the form of slush. It is an office procedure and local reactions vary from mild erythema to vesiculation, depending upon the method, time and pressure of application. It should be carried out by experienced personnel. The method is relatively painful since local anesthetic cannot be used, it is time consuming, and it may take a period of months and even years to bring about only a slight improvement of the scars,

so that the expected cosmetic improvement is not too good.

CHEMOTHERAPY

Many local applications have been invoked in order to exfoliate the epidermis and thereby improve the scars. The pits may be ringed with trichloracetic acid. This is a rather tedious and slow method of flattening scars and can only be employed when the lesions are few and discrete. The local reaction is severe and the method is not practical where there is extensive scarring.

Pure phenol may be applied over the entire face or to the affected sites: the reaction is severe or terrific, the skin turns white and burning is severe. The patient may temporarily have a faint feeling because of phenol absorption. The area at the site of application is immediately neutralized with a bland lotion. The patient is very uncomfortable for the next 48 hours, having a burning sensation. The skin turns absolutely black and then peels off in sheets in about 5 to 7 days. The patient may be incapacitated for 10 days.

There are numerous other chemical methods that may be used. However, the former two are the most popular. Again, these chemotherapeutic measures are safe applications in experienced hands, but are still fraught with dangers.

MECHANOTHERAPY

Abrasion of the skin by means of mechanical therapy results in improvement of the scars. The two popular methods employed are sandpapering and planing with a stainless steel wire brush. The sandpapering technique requires hospitalization and a general anesthetic is employed.

Our plastic planing method is a modification of the Kurtin technique utilizing a planing device, and is an office procedure. The patient, usually accompanied by a friend or relative, is given 50 mg. of Dermerol hydrochloride to allay any anxiety. An assistant sprays ethyl chloride on the operative site for local anesthesia and areas about the size of a silver dollar are abraded, the depth depending upon the judgment of the operator, until the entire face is completed. The face is dressed with dry gauze and removed when the patient returns home. Typewritten instructions are given to the patient regarding post-operative care. The procedure is painless and the improvement varies according to the depth of the abrasion. The process may be repeated at 3 month intervals if necessary, depending upon the discretion of the operator. It is a safe technique which offers the patient a ray of hope for the improvement of their acne scars.

SUMMARY AND CONCLUSION

We have attempted to broach the most common and important dermatologic procedures in the management of acne. The need for early and proper treatment is emphasized in order to prevent scarring. Education of parents and patients, together with a rational therapeutic approach will result in bringing this disease under control in the majority of instances.

817 West Street

REFERENCE

1. Crawford, G. M.: Treatment of Acne: A Symposium, Modern Medicine Annual, (1951) page 315-330.

Correction

In *The Journal* for December, 1954, on page 300, in the excellent article by Dr. O. J. Pollak on "Electrophoretic Fractionation of Serum Glucoproteins" his Figure 1 was printed upside down. *The Journal* regrets exceedingly this unfortunate happenstance and will do all it can to prevent a recurrence of such a mistake. Proper corrections are being made in the author's reprints.

+ Editorials +

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WATCHWORD FOR '55

No better presentation of the menace to medicine, in certain phases, has come to our attention than the December editorial in *Arizona Medicine*. Read it, as follows:

CAUTION?

THE POIGNANT cover of "Look", October 5th, 1954, with the caption, "Are Your Doctor Bills Padded?" must have been great for sales. The article by the same title discloses nothing about the padding of doctor's bills, except the last paragraph which refers the readers, if they suspect padding, to their Medical Society's Grievance Committee. The text of "Are Your Doctor's Bills Padded?" can be classed with others, to cite a few: "The Doctor's Conspiracy In Silence," "Why Some Doctors Should Be In Jail," "Patients For Sale," "Watch It, Doc!" "How Much Should Your Doctor Charge?".

Again, we must have been ranked high because "dirt" is more salable if it incriminates the renown. If the public and our profession are to be benefitted by such an expose, we would be the last to withhold our plaudits. However, if the whole medical profession is discredited by the repetitious exposure of the few erring doctors, and the prejudicial presentation of subject matter proves to be a cold war of class-against-class; then our present society shall crumble. Such a method has been proven effective by Hitler, Mussolini, Lenin and Stalin. Those who perpetrate such licentious implications should be repeatedly reminded of Lenin's statement when he proclaimed socialized medicine to be "the keystone of the arch of the Socialist state."

"Are Your Doctor Bills Padded," true to form, is biased by incomplete statements, quotation of the exceptions, its narrow range of comparisons, and by inference. It implies to its readers that the higher cost of medical care is unique and disproportionate, which a full recitation of the facts would belie. If not wholly for their sales value, these unqualified statements must have been made to soften the medical profession for something to come in the future.

The author implies that it is antisocial that a few families spend a high per cent of their income to protect their health — their most valuable asset, whereas, he does not mention what should be done to help those who lose all their worldly possessions by Acts of God. He compares our earnings to those of college professors and factory workers and by quotations, challenges that the discrepancies in incomes are unjustified. He fails to criticize our social system which condones individuals becoming wealthy from profits made by the sale of the essentials of life such as food, even though part of their profits come from those who are so poor that the medical profession renders them free medical care. Nor does he mention our way of life which allows people to become wealthy by unearned incomes from exploitation of our natural resources — or the sale of land and houses necessary for sheltering the poor.

When the medical profession is conquered our social planners will then turn and destroy other essential segments of our social structure. It is to be lamented that for "sales" many of our "popular writers" are blinded, and are no more aware that they are undermining their own palaces, than are their readers who make their sales possible.

What are the present facts as regards Governmental Medicine? We are closer than most realize. The 1954-55 budget of our government for health is over two billion dollars. This is eight per cent above the preceding year and is sixteen per cent of the total private public expenditures for health. In what other socialistic venture has our government gone so far?

We have just returned the balance of power of the legislative branch of our government to a party which has favored the extension of all governmental regulations in all fields. Will their trends of the past be enlarged? Will Oscar Ewings return? We must be alert; we must be cautious in our own affairs; we must be cautious and determined that those who represent us do not destroy our social system. We must keep ourselves, the public, and our legislators informed as to what is good for all.

People, including our representatives in government, learn and are impressed by what they read — so let us furnish them with something to read and not let them form their opinion from the too frequently biased articles which appear on our news stands or yes, even some of those which come from our government presses. It is the responsibility of all of us to diseminate pertinent information. Do not entrust this decisive job to any selected group.

DELAWARE STATE BOARD OF HEALTH

POLICY FOR LABORATORY OPERATION

The State Board of Health Laboratory will limit its work to laboratory tests of public health interest and will include case-finding tests for tuberculosis and cancer. No clinical laboratory procedures, such as blood counts, hemoglobin determinations, blood chemistry, or other tests of clinical significance, will be made by Board of Health personnel.

WHO MAY SUBMIT SPECIMENS FOR EXAMINATION:

1. Licensed physicians.
2. Public Health Officers.
3. Medical Officers in Federal Service.
4. Attorney General.

REPORTS

Reports will be submitted to licensed physicians and recognized clinics only, except reports on water, food, milk and similar substances may be transmitted to the municipality, business, or person responsible for maintaining standards which apply to such specific items.

TESTS WHICH THE LABORATORY WILL PERFORM:

1. **Serological and spinal fluid tests for syphilis.**
2. **Cultures of:**
   a. *blood* for typhoid, brucellosis, salmonellosis, shigella.
   b. *urine* for typhoid, shigella organisms, salmonella group.
   c. *feces* for typhoid, salmonella, shigella.
   d. *pus* for gonorrhea.
   e. *sputum* and *nasopharyngeal* secretion for diphtheria, b. hemolytic streptococcus (scarlet fever, septic sore throat), Vincent's infection, tuberculosis, and fungus infection.
3. **Agglutination or Precipitation tests:**
   a. Typhoid - Salmonella
   b. Undulant Fever
   c. Tularemia, Shigella
   d. Typhus
   e. Rocky Mountain Spotted Fever
   f. Rickettsial diseases
   g. Heterophil antibodies
4. **Complement Fixation tests:**
   a. Syphilis
   b. Gonorrhea
5. **Water:**
   a. Presumptive test for coliform.
   b. Confirmed test for coliform.
   c. Organism identification tests when indicated.
6. **Shellfish:**
   a. Presumptive test for coliform.
   b. Coliform counts.
   c. Plate counts.
   d. Organism identification.
7. **Milk and Dairy Products:**
   a. Plate counts.
   b. Breed counts.
   c. Coliform counts.
   d. Test for completeness of pasteurization.
   e. Undulant fever identification tests.
   f. Butter fat (when indicated).
   g. Identification tests if indicated.
8. **Rabies:**
   a. Examination for negri bodies.
   b. Animal inoculation.
9. **Urine:**
   a. Routine test for prenatal midwife cases.
10. **Food:**
    a. Routine test for contamination.
11. **Parasitology:**
    a. Body discharges for enteric parasite identification.
12. **Cystology:**
    a. Smears for malignant type cells by Papanicolaou method.
13. **Biopsies:**
    These are not performed in our lab for indigent cases, but are sent to qualified pathologists.

14. **Evaluation tests:**
For other labs doing serological tests for syphilis.

15. **Bacteriological Smears:**
For tentative identification of organisms.

NOTE: The State Board of Health reserves the right to perform other tests of public health interest when indicated.

1955 Delaware March of Dimes Against Polio

The polio attack rate in Delaware last year according to provisional reports was about the same as the national average for the year 1954. Nationwide, the number of cases reported in 1954 was the third highest on record.

The highest polio epidemic year in Delaware during recent times was in 1952, when 117 polio cases were reported. It is impossible to predict when and where polio epidemics will strike, which underlines the need for more effective control measures.

Evaluation of the Salk vaccine, administered to 440,000 U. S. children, in the largest medical experiment of its kind ever conducted, is now in progress. Announcement of the vaccine's effectiveness will be made in the Spring of 1955.

During the field trials last Spring about 2,400 children in the state of Delaware were inoculated with the Salk vaccine.

It is hoped that Delaware physicians will support the 1955 March of Dimes as enthusiastically as approximately 20,000 physicians throughout the United States cooperated in the 1954 vaccine field trials sponsored by the National Foundation for Infantile Paralysis.

This year the March of Dimes must do a bigger job than ever before. It must raise $64,000,000 — because $9,000,000 is needed to purchase vaccine, $2,700,000 for scientific research, $2,900,000 for professional education, and at least $29,900,000 for patient aid, including hospitalization. The March of Dimes has expended $203,600,000 in patient aid since 1938.

For science and humanity, give generously to the 1955 March of Dimes in January. Let your patients and friends know that the March of Dimes fights wisely, economically and effectively against the polio threat.

It is certain that tuberculosis is not an inescapable component of human society. It is always the result of gross defects in social organization and in the management of individual life. It is truly a social sin which can and must be stamped out. Rene J. Dubos, Ph.D., Am. Rev. Tuberc., July, 1953.

One of the greatest benefits of the mass survey has been to demonstrate to both the medical profession and the public the existing high prevalence of tuberculosis and the need for a continuous, efficient, case-finding program in each community. George Jacobson, M.D., and Denis C. Adler, M.D., Am. Rev. Tuberc., June, 1954.

Various health problems, which seem unrelated, actually are closely related. If the farmers in an area are all sick with malaria at harvest time, famine results. The lowered resistance of the starving population paves the way for more rapid spread of tuberculosis. Both malaria and tuberculosis result in lowered economic standards. Substandard housing and overcrowding follow and in turn contribute further to the development of tuberculosis. Extensive dental caries will result in malnutrition, which in turn may produce greater susceptibility to tuberculosis . . . the substandard living conditions resulting in part from such diseases cause discontent, frustration, and desperation—fertile soil for the growth of communism. James E. Perkins, M.D., NTA Bulletin, Sept., 1954.

A home care program for the tuberculous rests on the assumption that an intelligent, cooperative patient, partially restored to health, can be treated for part of his illness at home to his ultimate benefit. Editorial, GP, Jan., 1954.

IRA A. B. ALLEN, M.D.

Dr. Ira A. B. Allen, of Seaford, died on December 13, 1954, following a long period of illness. He was 75 years of age. He was born in Seaford on January 29, 1879, the son of the late John Wesley and Ida D. (Lloyd) Allen.

Dr. Allen received his early education in the public schools of Seaford and at the Pennsylvania State Normal School at West Chester, Pa. After teaching school for three years, he entered the University of Maryland Medical School, from which he graduated in 1905. His interneship was served in the Franklin Square Hospital, Baltimore.

After practicing for a short time in West Virginia, he located in Marion Station, Md. There he established a private hospital which subsequently consolidated with the McCready Memorial Hospital in Crisfield. He was for many years a member of the staff of the hospital.

In 1924 he completed a post-graduate course in surgery at the Post-graduate Hospital in New York City. Following this he moved to his old home in Seaford where he has been in active practice until illness made necessary his retirement.

He was a member of the Sussex County Medical Society and Medical Society of Delaware and of the American Medical Association.

He was a member of Chesapeake Lodge, No. 147, Free and Accepted Masons of Crisfield, Md., and was a life-long member of the Woodland Methodist Church.

For several years he was a member of the Board of Directors of the First National Bank of Seaford.

During World War I and II he enlisted in the Medical Service Corps of the Council of National Defense.

On December 18, 1901 Dr. Allen married the former Bathana Lowe, of Galestown, Md., who survives him. He is also survived by two children: Mrs. E. Cranston Riggin, of Baltimore, Md., and Dr. I. Benson Allen, of Seaford; two grandchildren, Mr. E. Cranston Riggin, Jr., and Miss Phyllis Lynn Allen; two sisters, Mrs. Samuel G. Smith and Mrs. G. Edwin Phillips, of Seaford.

Funeral services were conducted from his late home on December 16, 1954.

BOOK REVIEW

RECENT ADVANCES IN CARDIOVASCULAR PHYSIOLOGY AND SURGERY: A Symposium Presented by the Minnesota Heart Association and the University of Minnesota. Pp. 132. Paper, $1.00.

This booklet is a transcription of the papers and discussions at the symposium on cardiac physiology and surgery in September 1953. Your reviewer was privileged to attend this meeting and can report first-hand that it would be difficult, if not impossible, to assemble a more authoritative group of experts on the subject. The Stockholm group was well represented, in addition to leaders from Germany, Canada, and the United States.

The first three sessions dealt mostly with chemistry and experimental physiology and, while valuable, are of minor clinical interest. The fourth session (differential diagnosis of various defects by physiologic means) and the last two sessions (recent advances in surgical treatment of heart disease) are invaluable to the clinician.

This booklet should be in the library of any physician or surgeon who has any contact whatsoever with the diagnosis or treatment of heart disease.

Dramamine's® Effect in Vertigo

Dramamine has become accepted in the control of a variety of clinical conditions characterized by vertigo and is recognized as a standard for the management of motion sickness.

Vertigo, according to Swartout, is primarily due* to a disturbance of those organs of the body that are responsible for body balance. When the posture of the head is changed, the gelatinous substance in the semi-circular canals begins to flow. This flow initiates neural impulses which are transmitted to the vestibular nuclei. From this point impulses are sent to different parts of the body to cause the symptom complex of vertigo.

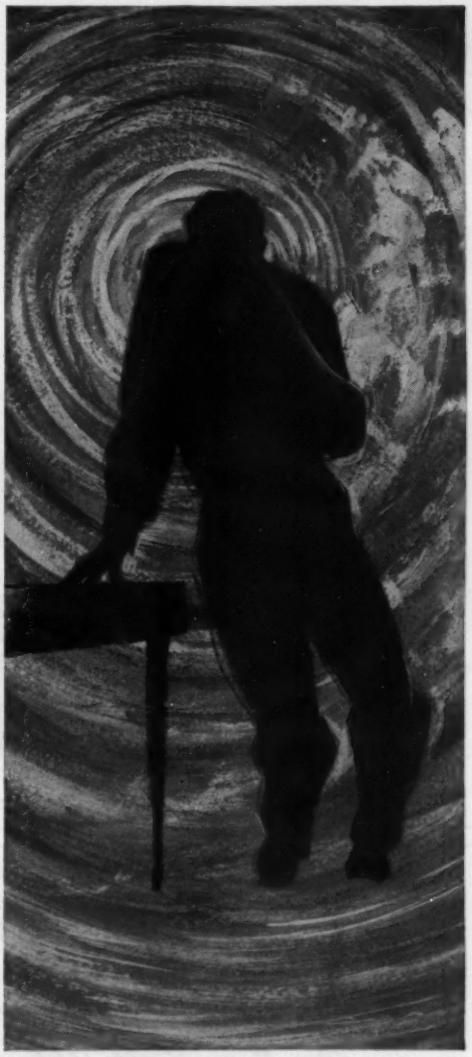
Some impulses reach the eye muscles and cause nystagmus; some reach the cerebellum and skeletal muscles and righting of the head results; others activate the emetic center to result in nausea, while still others reach the cerebrum making the person aware of his disturbed equilibrium. *Vertigo may be caused by a disease or abnormal stimuli of any of these tissues involved in the transmission of the vertigo impulse, including the cerebellum and the end organs.*

A possible explanation of Dramamine's action is that it depresses the overstimulated labyrinthine structure of the inner ear. Depression, therefore, takes place at the point at which these impulses, causing vertigo, nausea and similar disturbances, originate. Some investigators have suggested that Dramamine may have an additional sedative effect on the central nervous system.

Repeated clinical studies have established Dramamine as valuable in the control of the symptoms of Ménière's syndrome, the nausea and vomiting of pregnancy, radiation sickness, hypertension vertigo, the vertigo of fenestration procedures, labyrinthitis and vestibular dysfunction associated with antibiotic therapy, as well as in motion sickness.

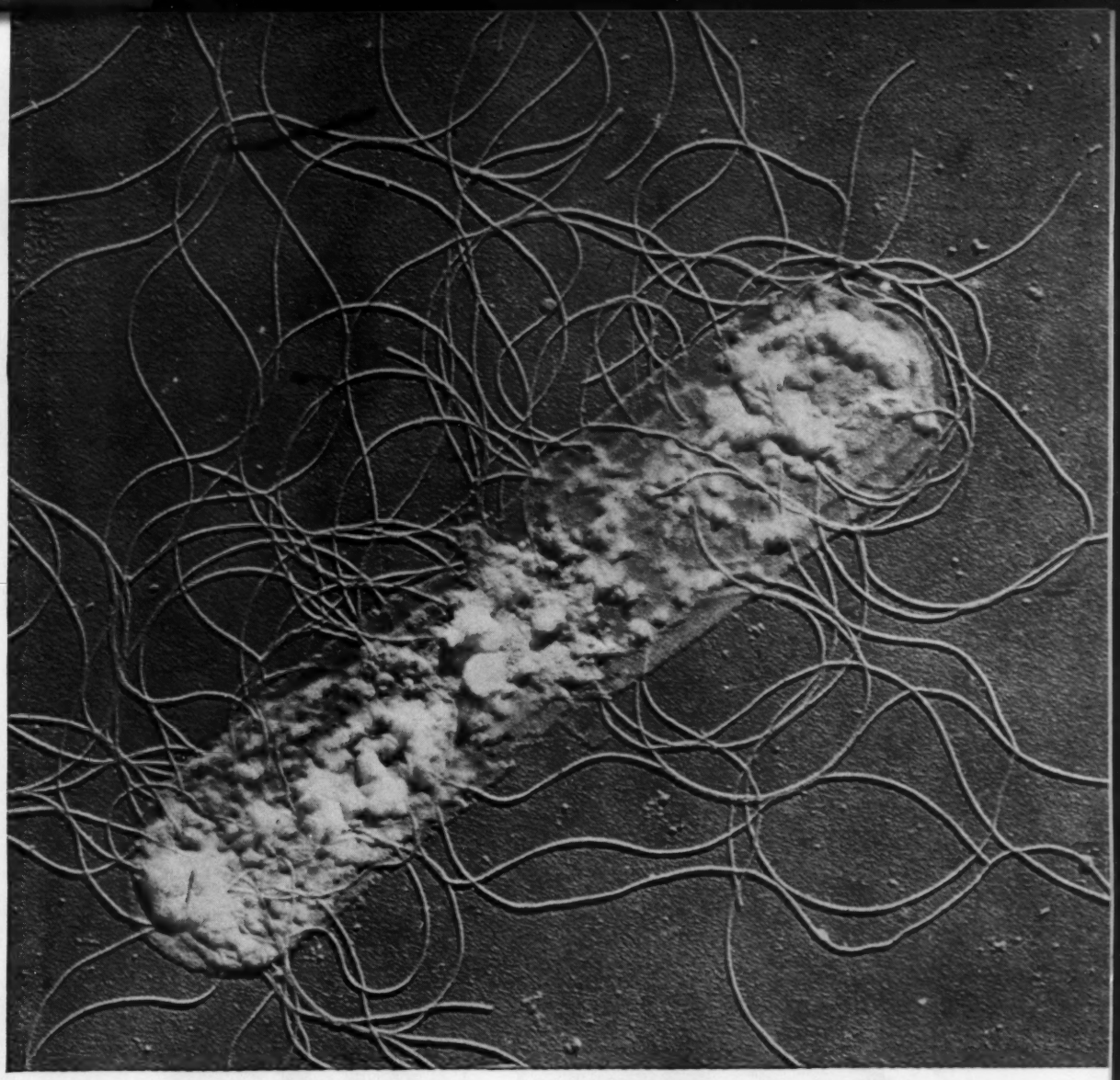
Any of these conditions in which Dramamine is effective may be classed as "disease or abnormal stimuli"* of the tissues including the end organs (gastrointestinal tract, eyes) and their nerve pathways to the labyrinth.

Dramamine (brand of dimenhydrinate) is supplied in tablets of 50 mg. and liquid (12.5 mg. in each 4 cc.). It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.



The site of Dramamine's action is probably in the labyrinthine structure.

*Swartout, R., III, and Gunther, K.: "Dizziness:" Vertigo and Syncope, GP *8*:35 (Nov.) 1953.



ELECTRON PHOTOMICROGRAPH COURTESY R.C.A. LABORATORIES

Proteus vulgaris 29,000 X

Proteus vulgaris is a Gram-negative organism commonly involved in

urinary tract infections • septicemia

peritonitis following low perforation of the gut.

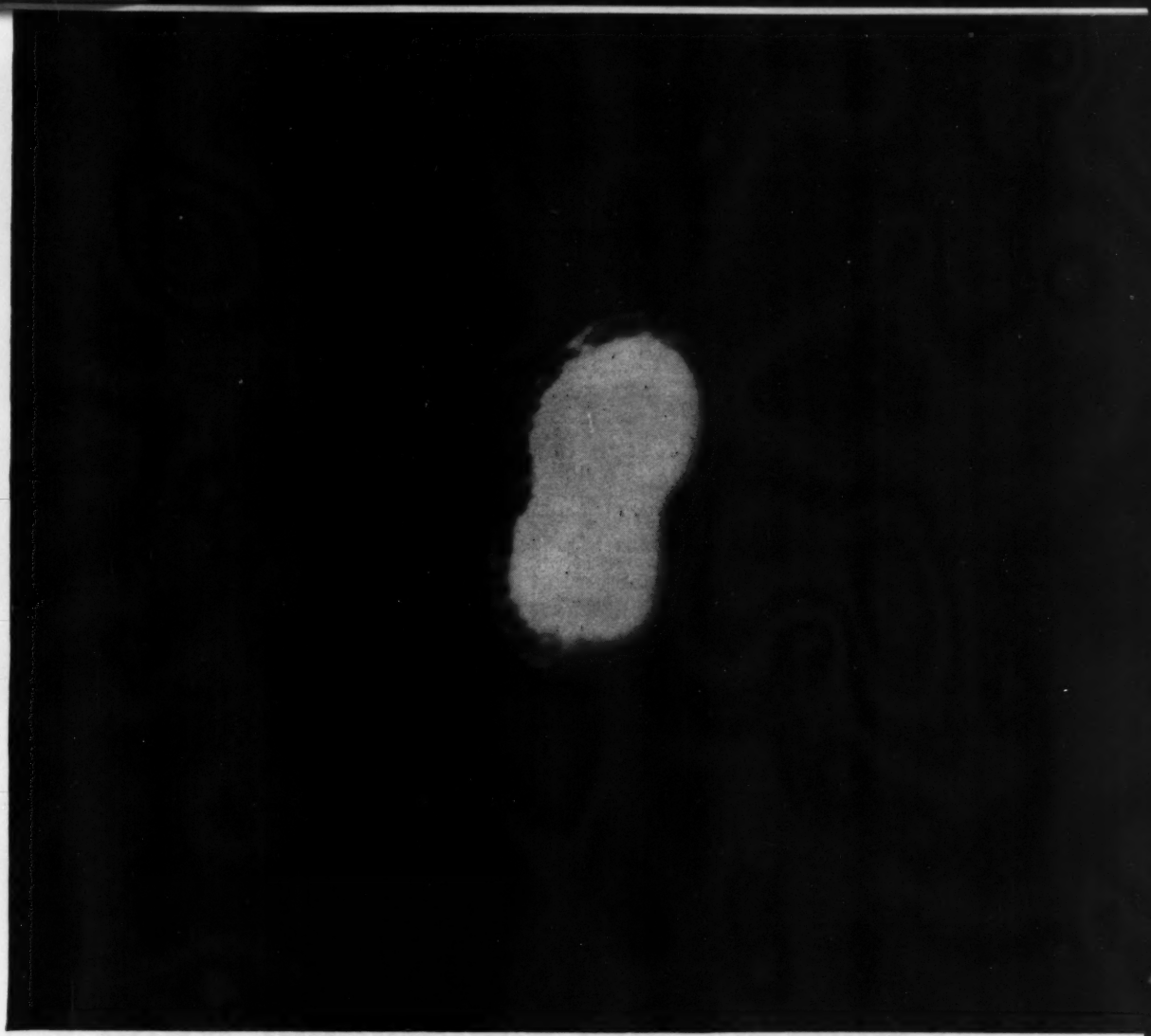
It is another of the more than 30 organisms susceptible to

PANMYCIN*

100 mg. and 250 mg. capsules

Upjohn

*TRADEMARK, REG. U. S. PAT. OFF.



ELECTRON PHOTOMICROGRAPH

Diplococcus pneumoniae 44,000 X

Diplococcus pneumoniae (Streptococcus pneumoniae) is a Gram-positive organism commonly involved in

lobar—and bronchopneumonia • chronic bronchitis • mastoiditis • sinusitis otitis media • and meningitis.

It is another of the more than 30 organisms susceptible to

PANMYCIN*

100 mg. and 250 mg. capsules

Upjohn

*TRADEMARK, REG. U. S. PAT. OFF.

Provides a protein margin of safety

- **for greater nitrogen retention**
- **for firmer muscle mass**

LIQUID

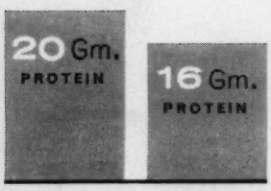
POWDERED

LACTUM

NUTRITIONALLY SOUND FORMULA FOR INFANTS

In the bottle-fed infant, a higher protein intake, with greater nitrogen retention, results in firmer muscle mass, better tissue turgor and better motor development.[1] A protein intake that does not maintain positive nitrogen balance "cannot be considered optimal or even safe for any length of time."[2]

During the first year of life, the infant's nourishment is derived primarily from his formula. Hence it is especially important that the formula be generous in protein. The usual Lactum® feedings provide 2 Gm. protein per pound of body weight—25% more than the Recommended Daily Allowance of 1.6 Gm. per pound (3.5 Gm. per kilogram).



Lactum formula for a 10 lb. infant

Recommended Daily Allowance for a 10 lb. infant

1. Jeans, P. C., in A.M.A. Handbook of Nutrition, Philadelphia, Blakiston, 1951, pp. 275-298. 2. Stare, F. J., and Davidson, C. S., in The Proteins, American Medical Association, 1945.

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